

Fourth Edition

Learning and Memory

From Brain to Behavior



Mark A. Gluck

Eduardo Mercado

Catherine E. Myers

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Description

The front cover

Learning and Memory

FROM BRAIN TO BEHAVIOR

Fourth Edition

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To my nifty nieces, Mandy (22) and Kamila (19): Over the course of four editions you have grown—both in real life and as the fictional characters herein—from darling little girls to delightful young ladies.

M. A. G.

In loving memory of Winnie Holsenbeck, who knew that learning had more to do with finding snakes under rocks than with answering teachers' questions.

E. M. III

To Moira, our “fourth author” and guiding spirit, with gratitude and respect for her talents, industry, and good humor throughout this project.

C. E. M.

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Mark A. Gluck is a Professor of Neuroscience and Public Health at Rutgers University–Newark and Director of the Rutgers Aging and Brain Health Alliance. His research focuses on the cognitive, computational, and neural bases of learning and memory; on the consequences of memory loss due to aging, posttraumatic stress disorder, and Alzheimer’s disease; and on how sleep and exercise can improve cognition and brain health. His international collaborations have included research in England, Hungary, China, Turkey, Israel, Egypt, and the Palestinian West Bank. Locally, he is active in community-engaged research in and around Newark, New Jersey. Gluck is co-author, with Catherine E. Myers, of *Gateway to Memory: An Introduction to Neural Network Modeling of the Hippocampus and Learning* (MIT Press, 2001), is co-editor of three other books, and has published more than 130 peer-reviewed journal articles. In 1996, he was awarded an NSF Presidential Early Career Award for Scientists and Engineers by President Bill Clinton and also received the American Psychological Association (APA) Distinguished

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her research is available at www.cemyers.com.

BRIEF CONTENTS

Preface

INTRODUCTORY MODULE

CHAPTER 1 Fundamental Themes in the Psychology of Learning and Memory

CHAPTER 2 The Neuroscience of Learning and Memory

LEARNING MODULE

CHAPTER 3 Habituation, Sensitization, and Familiarization: Learning About Repeated Events

CHAPTER 4 Classical Conditioning: Learning to Predict Significant Events

CHAPTER 5 Operant Conditioning: Learning the Outcome of Behaviors

CHAPTER 6 Generalization, Discrimination Learning, and Concept Formation

MEMORY MODULE

CHAPTER 7 Episodic Memory and Semantic Memory: Memory for Events and for Facts

CHAPTER 8 Skill Memory: Learning by Doing

CHAPTER 9 Working Memory and Cognitive Control

INTEGRATIVE TOPICS MODULE

CHAPTER 10 Emotional Influences on Learning and Memory

CHAPTER 11 Social Learning and Memory: Observing, Interacting, and Reenacting

CHAPTER 12 Development and Aging: Learning and Memory Across the Lifespan

Glossary

References

Name Index

Subject Index

CONTENTS

Preface

INTRODUCTORY MODULE

CHAPTER 1 Fundamental Themes in the Psychology of Learning and Memory

Linking Ideas in the Mind

William James and Memory Networks

Learning and Memory in Everyday Life: Top 10 Tips for a Better Memory

Ivan Pavlov's Conditioning Studies

Edward Thorndike and the Law of Effect

Nature Versus Nurture

Descartes and Dualism

John Locke and the Blank Slate

John Watson's Behaviorism

B. F. Skinner's Radical Behaviorism

Comparing Humans to Other Animals

Charles Darwin and the Theory of Natural Selection

The Neo-Behaviorism of Edward Tolman

Formal Models and Universal Laws

Hermann Ebbinghaus and Human Memory Experiments

Learning and Memory in Everyday Life: What If You Could Form No New Memories?

Clark Hull and Mathematical Models of Learning

W. K. Estes and Mathematical Psychology

George Miller and Information Theory

The Connectionist Models of David Rumelhart

Synthesis

CHAPTER 2 The Neuroscience of Learning and Memory

Learning and Memory in Everyday Life: Top Five Tips for Faster Forgetting

Structural Plasticity in Nervous Systems

Brains

The Human Brain

Imaging Brain Structure

Neurons and Glia

How Experience Changes Brain Structure

Imprinting

Environmental Enrichment

On-the-Job Training

Hebbian Learning

Functional Properties of Learning and Memory Systems

What Brains Do

Incoming Stimuli: Sensory Pathways into the Brain

Outgoing Responses: Motor Control

Observing Brains in Action

How Remembering Changes Brain Activity

Finding and Manipulating Memories

Looking for Memories

Searching for Memories in the Cerebral Cortex

The Synapse: Where Neurons Connect

Long-Term Potentiation

How Researchers Change Brain Activity

Electromagnetic Stimulation of Neurons

Biochemical Control of Brain States

Learning and Memory in Everyday Life: Can a Pill Improve Your Memory?

Synthesis

LEARNING MODULE

CHAPTER 3 Habituation, Sensitization, and Familiarization: Learning About Repeated Events

Behavioral Processes

Recognizing and Responding to Repetition

General Features of Habituation

Stimulus Specificity and Dishabituation

Factors Influencing the Rate and Duration of Habituation

Learning and Memory in Everyday Life: Sex on the Beach

General Features of Sensitization

Dual Process Theory

Opponent Process Theory

The What and Where of Learning from Exposure

Recognizing Familiar Objects

Priming

Perceptual Learning

Learning and Memory in Everyday Life: Unconscious Racial Bias

Spatial Learning

Brain Substrates

An Invertebrate Model System

Habituation in *Aplysia*

Sensitization in *Aplysia*

Perceptual Learning and Cortical Plasticity

Cortical Maps of Sensations

Cortical Plasticity During Development

Cortical Changes in Adults After Exposure

Hippocampal Involvement in Spatial Learning and Familiarity

Identifying Places

Recognizing Familiar Objects

Clinical Perspectives

Rehabilitation After Stroke: Habituation Gone Awry

Sensitization to Stress in Anxiety and Depression

[Human–Machine Interfaces: Regaining Sensory Modalities Through Perceptual Learning](#)

[Synthesis](#)

[**CHAPTER 4 Classical Conditioning: Learning to Predict Significant Events**](#)

[**Behavioral Processes**](#)

[Basic Concepts of Classical Conditioning](#)

[How Pavlov Conditioned Dogs to Salivate](#)

[Appetitive Conditioning](#)

[Aversive Conditioning](#)

[Mammalian Conditioning of Motor Reflexes: Eyeblink Conditioning](#)

[Learning a New Association](#)

[Refining the Basic Principles](#)

[Conditioned Compensatory Responses](#)

[What Cues Can Be CSs or USs?](#)

[Extinguishing an Old Association](#)

[Compound Conditioning and Overshadowing](#)

[Error Correction and the Modulation of US Processing](#)

[The Informational Value of Cues](#)

[Kamin’s Blocking Effect](#)

[The Rescorla–Wagner Model of Conditioning](#)

[Error-Correction Learning](#)

[Associative Weights and Compound Conditioning](#)

[Using the Rescorla–Wagner Model to Explain Blocking](#)

[Influence of the Rescorla–Wagner Model](#)

[Error Correction in Human Category Learning](#)

[Stimulus Attention and the Modulation of CS Processing](#)

[An Attentional Approach to Stimulus Selection](#)

[An Attentional Explanation of Latent Inhibition](#)

[Other Determinants of Conditioning](#)

[Timing](#)

[Associative Bias](#)

Brain Substrates

[Conditioning of Motor Reflexes in the Mammalian Brain](#)

[Electrophysiological Recording in the Cerebellum](#)

[Brain Stimulation as a Substitute for Behavioral Training](#)

[Impaired Conditioning Following Cerebellar Damage](#)

[Error Correction Through Inhibitory Feedback](#)

[The Hippocampus in CS Modulation](#)

[Invertebrates and the Cellular Basis of Learning](#)

[Presynaptic Versus Postsynaptic Changes During Learning](#)

[Long-Term Structural Changes and the Creation of New Synapses](#)

Clinical Perspectives

[Tolerance to Addictive Drugs](#)

[Reducing the Need for Medication or Reducing Its Side Effects](#)

[Learning and Memory in Everyday Life: Extinguishing a Drug Habit](#)

Synthesis

CHAPTER 5 Operant Conditioning: Learning the Outcome of Behaviors

Behavioral Processes

[The “Discovery” of Operant Conditioning](#)

[Classical Versus Operant Conditioning](#)

[Free-Operant Learning](#)

[Learning About Stimuli and Responses](#)

[Discriminative Stimuli](#)

[Responses](#)

[Shaping](#)

[Chaining](#)

[Learning About Outcomes](#)

[Learning and Memory in Everyday Life: Drug-Detecting Dogs](#)

[Primary Reinforcers](#)

[Secondary Reinforcers](#)

[Punishers](#)

[Differential Reinforcement of Alternative Behaviors](#)

[Learning and Memory in Everyday Life: The Problem with Punishment](#)

[Putting It All Together: Building the \$S^D \rightarrow R \rightarrow O\$ Association](#)

[Timing Affects Learning](#)

[Outcomes Can Be Added or Subtracted](#)

[Reinforcement Need Not Follow Every Response](#)

[Reinforcement Schedules](#)

[Choice Behavior](#)

[The Matching Law](#)

[Behavioral Economics](#)

[Delayed Reinforcement and Self-Control](#)

[Reciprocity and Altruism](#)

[Brain Substrates](#)

[The Dorsal Striatum and Stimulus–Response \(\$S^D \rightarrow R\$ \) Learning](#)

[The Orbitofrontal Cortex and Learning to Predict Outcomes](#)

[Predicting Specific Outcomes](#)

[Delayed Reinforcement and Self-Control](#)

[Mechanisms of Reinforcement Signaling in the Brain](#)

[“Wanting” and “Liking” in the Brain](#)

[Dopamine: How the Brain Signals “Wanting”?](#)

[Endogenous Opioids: How the Brain Signals “Liking”?](#)

[How Do “Wanting” and “Liking” Interact?](#)

[Punishment Signaling in the Brain](#)

[The Insula: How Much Does It Hurt?](#)

[The Dorsal Anterior Cingulate Cortex: What to Do About It?](#)

[Clinical Perspectives](#)

[Drug Addiction](#)

[Behavioral Addiction](#)

[Treatments for Addiction](#)

[Synthesis](#)

CHAPTER 6 Generalization, Discrimination Learning, and Concept Formation

Behavioral Processes

[Generalization: When Similar Stimuli Predict Similar Outcomes](#)

[Generalization as a Search for Similar Consequences](#)

[The Challenge of Incorporating Similarity into Learning Models](#)

[Learning and Memory in Everyday Life: How Does Amazon.com Know What You Want to Buy Next?](#)

[Shared Elements and Distributed Representations](#)

[Discrimination Learning and Stimulus Control: When Similar Stimuli Predict Different Outcomes](#)

[Discrimination Learning and Learned Specificity](#)

[Learning and Memory in Everyday Life: Sleep Better Through Stimulus Control](#)

[Errorless Discrimination Learning and Easy-to-Hard Transfer](#)

Beyond Similarity

[Sensory Preconditioning: Co-occurrence and Stimulus Generalization](#)

[Acquired Equivalence: Novel Similar Predictions Based on Prior Similar Consequences](#)

[Negative Patterning: When the Whole Means Something Different Than the Parts](#)

[Variability in Training and the Generalization of Knowledge and Skills](#)

[Concept Formation, Category Learning, and Prototypes](#)

[Emergence of Concepts Through Discrimination Learning](#)

[Prototypes and the Structure of Natural Categories](#)

[Stereotypes, Discrimination, and Racism in Generalizations About Other People](#)

[How Do People Learn Stereotypes?](#)

[How Do People Interpret and Use Stereotypes?](#)

[How Can We Distinguish Between Appropriate and Inappropriate Uses of Stereotypes?](#)

Brain Substrates

[Cortical Representations and Generalization](#)

[Cortical Representations of Sensory Stimuli](#)

[Shared-Elements Models of Receptive Fields](#)

[Evidence of Primary Cortex Role in Generalization](#)

[Plasticity of Cortical Representations](#)

[Generalization and the Hippocampal Region](#)

[Effect of Damage to the Hippocampal Region](#)

[Modeling the Role of the Hippocampus in Adaptive Representation](#)

Clinical Perspectives

[Generalization Deficits in Schizophrenia](#)

[Acquired Equivalence in Schizophrenia](#)

[Other Studies of Generalization in Schizophrenia](#)

[Altered Generalization in Autism Spectrum Disorder](#)

Synthesis

MEMORY MODULE

CHAPTER 7 Episodic Memory and Semantic Memory: Memory for Events and for Facts

Behavioral Processes

[Features of Episodic Memory and Semantic Memory](#)

[What Distinguishes Episodic Memory from Semantic Memory?](#)

[Which Comes First: Episodic Memory or Semantic Memory?](#)

[Can Nonhumans Have Semantic Memory?](#)

[Can Nonhumans Have Episodic Memory?](#)

[Encoding New Memories](#)

[Mere Exposure to Information Does Not Guarantee Memory](#)

[Memory Is Better for Information That Relates to Prior Knowledge](#)

[Deeper Processing at Encoding Improves Recall Later](#)

[Retrieving Existing Memories](#)

[Encoding Specificity: Similar Conditions at Encoding and Retrieval](#)

[Transfer-Appropriate Processing: Similar Processing at Encoding and Recall](#)

[More Cues Mean Better Recall](#)

[Struggling \(and Even Failing\) to Remember Can Improve Memory](#)

[Learning and Memory in Everyday Life: Extraordinary Memorizers](#)

[When Memory Fails](#)

[Forgetting](#)

[Interference](#)

[Source Monitoring](#)

[False Memory](#)

[Memory Consolidation and Reconsolidation](#)

[The Consolidation Period](#)

[Reconsolidation](#)

[Metamemory](#)

[Learning and Memory in Everyday Life: Optimize Your Study Habits](#)

[Brain Substrates](#)

[Cortical Networks for Semantic Memory](#)

[Single Neurons Respond Selectively to Specific Stimuli or Categories](#)

[Neuronal Networks Encode Mental Representations](#)

[The Medial Temporal Lobes and Memory Storage](#)

[The Hippocampus and Encoding of New Episodic Memories in Humans](#)

[The Hippocampus and Encoding of Episodic-Like Memories in Nonhuman Animals](#)

[The Medial Temporal Lobes and Encoding of New Semantic Memories](#)

[Determining What Gets Stored](#)

[The Frontal Cortex](#)

[The Basal Forebrain](#)

Long-Term Storage and Retrieval

Retrograde Amnesia

Standard Consolidation Theory

Multiple Trace Theory

Evidence from Healthy Brains

The Role of Sleep

Learning While You Sleep

Clinical Perspectives

Learning and Memory in Everyday Life: The Cost of Concussion

Transient Global Amnesia

Functional Amnesia

Synthesis

CHAPTER 8 Skill Memory: Learning by Doing

Behavioral Processes

Features of Skill Memories

Similarities Between Perceptual-Motor Skills and Cognitive Skills

Which Comes First: Cognitive or Perceptual-Motor Skill Memory?

Can Nonhumans Have Cognitive Skill Memories?

Encoding New Memories

More Repetition Does Not Guarantee Improvement

Timing and Sequencing of Practice Matters

Skill Memories Are Often Formed Unconsciously

Becoming an Expert

Talent Blossoms with Practice

Learning and Memory in Everyday Life: Are Some Cognitive Skills Easier for Men Than for Women?

Retrieving Existing Memories

When Memory Fails

Brain Substrates

The Basal Ganglia and Skill Learning

Learning Deficits After Lesions

Neural Activity During Perceptual-Motor Skill Learning

Learning and Memory in Everyday Life: Are Video Games Good for the Brain?

Brain Activity During Cognitive Skill Learning

Cortical Representations of Skills

Cortical Expansion

Are Skill Memories Stored in the Cerebral Cortex?

The Cerebellum and Timing

Clinical Perspectives

Parkinson's Disease

Human-Machine Interfaces: Learning to Consciously Control Artificial Limbs

Synthesis

CHAPTER 9 Working Memory and Cognitive Control

Behavioral Processes

Transient Memories

Sensory Memory

Short-Term Memory

Working Memory

Baddeley's Working-Memory Model

The Phonological Loop

The Visuospatial Sketchpad

Is Working Memory a Place or a State?

Cognitive Control

Controlled Updating of Short-Term Memory Buffers

Goal Setting and Planning

Task Switching

Stimulus Selection and Response Inhibition

Are Working Memory and Cognitive Control the Keys to Intelligence?

Learning and Memory in Everyday Life: Give Your Working Memory a Break

Brain Substrates

Frontal-Lobe Anatomy and Consequences of Frontal-Lobe Damage

Behavior Changes Following Frontal-Lobe Damage

Deficits in Working Memory Following Frontal-Lobe Damage

Divisions of the Prefrontal Cortex

Studies of Frontal Brain Activity During Working-Memory Tasks

Mapping Executive Processing and Working Memory onto PFC Anatomy

Maintenance (Rehearsal) Versus Manipulation (Cognitive Control)

Visuospatial and Phonological–Verbal Working Memory

The Neural Bases of State-Based Accounts of Working Memory

Goal Abstraction and Frontal-Lobe Organization

Prefrontal Control of Long-Term Declarative Memory

Clinical Perspectives

The Prefrontal Cortex in Schizophrenia

Inefficient Prefrontal Cortical Systems in Schizophrenia

Dopamine and the Genetics of Schizophrenia

Attention-Deficit/Hyperactivity Disorder

Synthesis

INTEGRATIVE TOPICS MODULE

CHAPTER 10 Emotional Influences on Learning and Memory

Behavioral Processes

What Is Emotion?

Autonomic Arousal and the Fight-or-Flight Response

James–Lange Theory of Emotion

Cannon–Bard Theory of Emotion

Two-Factor Theory of Emotion

Assessing Emotion in Nonhuman Animals

Fear Responses Across Species

[Fight-or-Flight or Tend-and-Befriend?](#)

[Beyond Fear](#)

[Learning About Emotion-Evoking Stimuli: Focus on Fear](#)

[Conditioned Emotional Responses: Learning to Predict Danger](#)

[Conditioned Escape: Learning to Get Away from Danger](#)

[Conditioned Avoidance: Learning to Avoid Danger Altogether](#)

[Learned Helplessness](#)

[Learning and Memory in Everyday Life: “Immunizing” Against Learned Helplessness](#)

[Memory for Emotional Events](#)

[Emotion and Encoding of Memories](#)

[Emotion and Retrieval of Memories](#)

[Flashbulb Memories](#)

[Learning and Memory in Everyday Life: Truth or Consequences](#)

[Brain Substrates](#)

[Emotional Learning in the Brain](#)

[The Amygdala and Expression of Emotional Responses](#)

[Two Pathways into the Amygdala: “Fast-and-Rough” Versus “Slower-but-Accurate”](#)

[Where Emotional Responses Are Learned](#)

[Where Context Is Learned](#)

[Conscious Feelings and the Frontal Lobes](#)

[Emotional Memory in the Brain](#)

[Amygdala Activation Can Strengthen Memory Encoding](#)

[Stress Hormones Can Strengthen Memory Encoding](#)

[Stress Hormones Can Impair Recall](#)

[Learning and Memory in Everyday Life: How Much Stress Is Too Much?](#)

[Stress Hormones Can Either Strengthen or Impair Reconsolidation](#)

[Clinical Perspectives](#)

Phobias

What Causes Phobias?

Therapy for Phobias

Posttraumatic Stress Disorder

Predicting and Preventing PTSD

Therapy for PTSD

Predicting and Preventing PTSD

Synthesis

CHAPTER 11 Social Learning and Memory: Observing, Interacting, and Reenacting

Behavioral Processes

Copying What Is Seen

Social Learning Theory

Studies of True Imitation: Replicating Actions

Studies of Emulation: Reproducing Outcomes

Matching Actions Without Truly Imitating

Copying What Is Heard

Vocal Imitation: Reproducing Sounds

Learning and Memory in Everyday Life: Karaoke

Learning to Vocalize Without Imitating

Genes and Memes in Song Learning

Social Transmission of Information

Learning Through Social Conformity

Social Manipulation of Behavior

Learning and Memory in Everyday Life: Learning What to Like from Super Bowl Ads

Brain Substrates

Identifying Mirror Neurons

Mirror Neurons in Songbirds

Clinical Perspectives

[Imitative Deficits After Stroke](#)

[Autism Spectrum Disorder](#)

[Synthesis](#)

CHAPTER 12 Development and Aging: Learning and Memory Across the Lifespan

Behavioral Processes

[The Developing Memory: Infancy Through Childhood](#)

[Learning Before Birth](#)

[Conditioning and Skill Learning in Young Children](#)

[Development of Episodic Memory and Semantic Memory](#)

[Sensitive Periods for Early Learning](#)

[Imprinting](#)

[Sensitive Periods for Vision and Birdsong](#)

[Language Learning](#)

[Learning and Memory in Everyday Life: Teaching Babies Signs Before Speech](#)

[Adolescence: Crossing from Childhood into Adulthood](#)

[Maturation of Working Memory](#)

[Male–Female Differences in Learning and Memory](#)

[The Aging Memory: Adulthood Through Old Age](#)

[Working Memory in Aging Adults](#)

[Conditioning and Skill Learning in Aging Adults](#)

[Episodic Memory and Semantic Memory: Old Memories Fare Better than New Learning](#)

[Metamemory and Aging](#)

Brain Substrates

[The Genetic Blueprint Is Modified by Experience](#)

[Genetic Variation and Individual Differences in Learning Abilities](#)

[Selective Breeding and Twin Studies](#)

[Environmental Interactions](#)

[Epigenetics](#)

Learning and Memory in Everyday Life: Can Exposure to Classical Music Make Babies Smarter?

Neurons and Synapses in the Developing Brain

Early Overproduction of Neurons

Pruning of Synapses

Sensitive Periods for Neuronal Wiring

Brain Changes in Adolescence

Profound Changes in Prefrontal Cortex

Effects of Sex Hormones on Brain Organization

Effects of Circulating Sex Hormones on Brain Function

The Brain from Adulthood to Old Age

Localized Neuron and Synapse Loss

Loss of Synaptic Stability and Consolidation

Adult Neurogenesis: New Neurons for Old Brains?

Clinical Perspectives

Down Syndrome

Brain Abnormalities and Memory Impairments in Down Syndrome

Animal Models of Down Syndrome

Alzheimer's Disease

Progressive Memory Loss and Cognitive Deterioration

Plaques and Tangles in the Brain

Genetic Basis of Alzheimer's Disease

A Connection Between Down Syndrome and Alzheimer's Disease

Learning and Memory in Everyday Life: Can Mental Exercise Protect Against Alzheimer's Disease?

Synthesis

Glossary

References

Name Index

[Subject Index](#)

PREFACE

This fourth edition of *Learning and Memory: From Brain to Behavior* is based on the successful first three editions, published in 2008, 2014, and 2016 and also available in German, Spanish, and Korean translations. This new edition continues our approach of presenting a comprehensive, accessible, and engaging introduction to the scientific study of learning and memory. The earlier editions have proven the value of our *innovative, modular organization, which offers a highly flexible curriculum* suited to the many ways different teachers prefer to teach this material. Other features that have earned the book its place as a leader in the field are its

- *Extensive use of real-world examples*, concrete applications, and clinically relevant perspectives, emphasizing how basic research impacts everyday life and clinical practice
- *Strong focus on neuroscience* that nevertheless succeeds in being unintimidating and clear for students who have little or no previous knowledge of the subject
- *Integrated coverage* of learning and memory in nonhuman animals and humans alike
- *Consistent division of each of the core chapters into three sections*—“Behavioral Processes,” “Brain Substrates,” and “Clinical Perspectives”—giving readers an awareness of what to expect as the chapter proceeds and allowing teachers to assign reading selectively to better suit their individual syllabi
- *Thorough integration of topics across chapters*, highlighting connections between themes and concepts that arise repeatedly in different parts of the book
- *Engaging and conversational writing style and extensive four-color art program*
- *Integrated in-chapter and end-of-chapter pedagogy*, providing students with immediate practice and feedback to help them assess their own progress and understanding, organize and prioritize information, and use the book more effectively in general
- *Extensive support of teachers* through supplemental materials

New in the Fourth Edition

The fourth edition of *Learning and Memory: From Brain to Behavior* is the fruit of a thorough review and evaluation process aimed at retaining the popular strengths of the previous editions and improving on those strengths whenever possible. This has resulted in the addition of even more real-life examples that are meaningful to students; an increase in coverage of learning and memory research and use of brain-imaging results; expanded discussion of the role of genetics in producing individual differences in learning and memory; and insertion of new material on a range of relevant topics, including the role of sleep in memory. In great part, the changes in the book fall into four categories, listed here with representative examples.

Laying Foundations and Promoting Comprehension

- Complete revision of [Chapter 1](#). This previously chronological introduction to the field has a new, more economical presentation organized around themes and concepts to provide a clearer picture of how the historical work is directly related to modern research.
- Refinements in the discussion of classical conditioning, including more concise coverage of the Rescorla–Wagner model in [Chapter 4](#). Discussion of contingency learning has been moved to the Instructor’s Resource Manual.
- Revision of [Chapter 6](#), on generalization, discrimination learning, and concept formation, to increase accessibility and readability. The discussion of teaching pigeons to distinguish styles of art has been expanded, and advanced concepts have been moved to the Instructor’s Resource Manual, resulting in a chapter that is shorter and simpler overall, with less dependence on mathematics and formal modeling.
- Simplified discussion of basic theories of skill learning and memory ([Chapter 8](#)).
- Clarification of distinctions between working memory and short-term memory ([Chapter 9](#)).
- Reorganization and simplification of brain substrates discussions in [Chapter 10](#), on emotional influences on learning and memory.
- New discussion clarifying the concepts of vocal learning and vocal imitation ([Chapter 11](#)).
- Expanded coverage of sex differences in learning and memory, explaining how male–female effects observed “on average” (between-groups effects) are usually swamped by individual differences within a gender ([Chapter 12](#)).
- Improvements to the illustration program throughout the text, including clarifications in figures depicting brain imaging. Specific colors are applied consistently in drawings of presynaptic and postsynaptic neurons as well as to plots of research data in graphs.

- Addition of photographs and other figures throughout the text to increase the illustration of central concepts.

Helping Students See the Relevance of Subject Matter

- Additions to the real-world examples of habituation, dishabituation, and sensitization in [Chapter 3](#) and of operant conditioning in [Chapter 5](#).
- Discussion of classic taste aversion conditioning as a means of helping patients tolerate the side effects of chemotherapy ([Chapter 4](#)).
- New material on the importance of variability in training ([Chapter 6](#)) and on the claim that 10,000 hours of deliberate practice is necessary to become an expert ([Chapter 8](#)).
- A new box on using insights from memory research to optimize study habits ([Chapter 7](#)).
- Additional discussion of the role of sleep and of the possibility of using targeted memory reactivation during sleep to enhance memory ([Chapter 7](#)).
- Added material on how environmental enrichment affects development ([Chapter 12](#)).

Fine-Tuning the General Content Coverage

- Inclusion of the role of glia in learning and synaptic plasticity and of imprinting as an example of how experience changes brains ([Chapter 2](#)).
- Inclusion of statistical learning as a special case of learning from exposure ([Chapter 3](#)).
- Expanded discussion of behavioral economics, including new material on delayed discounting, self-control, and altruism ([Chapter 5](#)).
- Added discussion of theories of memory consolidation ([Chapter 7](#)).
- Expanded discussion of learning sets ([Chapter 8](#)).
- Added material on the “tend-and-befriend” concept in the coverage of “fight-or-flight” responses ([Chapter 10](#)).
- Expanded discussion of emulation as a social learning mechanism and of the use of “ghost displays” ([Chapter 11](#)).

Staying Up-to-Date

- Inclusion of the neural substrates of highly superior autobiographical memory ([Chapter 2](#)).
- Inclusion of new technologies for personal monitoring, such as use of electrodermal activity for measuring habituation and sensitization ([Chapter 3](#)).
- Inclusion of the newly defined Internet gaming disorder ([Chapter 5](#)).

- Inclusion of developments in functional neuroimaging, including high-resolution fMRI studies to show how “memory” patterns are encoded in the cortex ([Chapter 7](#)), and new information on how techniques of optogenetics are helping tease apart cause and effect in the brain substrates of emotional learning and memory ([Chapter 10](#)).
- Updates on the ongoing debates about the role of the hippocampus in semantic memory ([Chapter 7](#)) and about adult neurogenesis in humans ([Chapter 12](#)).
- Updated discussion of mirror neurons in imitation and song learning ([Chapter 11](#)).
- Updated overview of social learning theory ([Chapter 11](#)).
- New material on how sleep disruption may contribute to memory decline in aging ([Chapter 12](#)).

Signature Features Retained in the Fourth Edition

The original conception of a textbook with a unique, modular table of contents, a significant focus on neuroscientific underpinnings, integrated coverage of both human and animal research, strong attention to clinical applications, unequalled pedagogy and appeal for students, and extensive supplemental support remains unchanged.

Flexible, Modular Table of Contents

There are at least four different ways in which teachers choose to teach the subject matter presented in this textbook:

1. **Learning only.** Focusing on animal conditioning and behaviorist approaches; teachers may or may not include neuroscience perspectives.
2. **Memory only.** Focusing primarily on human memory and cognition; teachers include varying degrees of cognitive neuroscience perspectives.
3. **Learning, then memory.** Starting with basic learning phenomena such as habituation, sensitization, and associative conditioning—phenomena most extensively studied in animals—and progressing to the more complex facets of human memory. Neuroscience coverage, when included, begins with the most elemental building blocks of neurons and circuits and works up to the larger anatomical perspectives required by human memory studies.
4. **Memory, then learning.** Here, teachers start with the engaging and familiar subject of human memory, including its many failings and idiosyncrasies, topics that students usually find especially relevant and appealing. As the course progresses, teachers present material on how human memory is built up from basic processes that can be studied in greater precision in animal models. Neuroscience coverage begins with the very accessible and easily understood big-picture view of anatomical regions and their functional relevance and then works toward presenting the more detailed neuronal focus of studies that can be done invasively in animal preparations, especially studies of conditioning and other forms of associative learning.

With *Learning and Memory: From Brain to Behavior*, there is now available a single textbook that is sufficiently modular in its overall structure and in the execution of individual chapters to accommodate these and any of the other common approaches to teaching this material.

How can one textbook suit every teaching approach? To accomplish this feat, we have divided the book into four multichapter modules:

- The **Introductory Module** is the natural starting point for all courses; teachers can assign either or both of two introductory chapters: one a conceptual and historical overview of the study of psychology and behavior, and the other an introduction to the neuroscience of learning and memory.
- The heart of the book consists of two “parallel” modules, the **Learning Module** and the **Memory Module**. These can be covered alone (for those teachers who wish to teach only learning or only memory) or in either order, allowing for a learning-then-memory syllabus or a memory-then-learning syllabus. Each of these modules is a self-contained collection of chapters, neither of which assumes that the student has read the other module. The Learning Module has four chapters, covering basic exposure-driven learning mechanisms; classical conditioning; operant conditioning; and, finally, generalization, discrimination, and similarity. The Memory Module has three chapters, covering episodic and semantic memory, skill memory, and working memory and cognitive control.
- The final module of the book, the **Integrative Topics Module**, consists of three optional stand-alone chapters (so that any subset of the three can be assigned), covering emotional learning and memory, social learning and memory, and lifespan changes in learning and memory, from prenatal development to old age.

Given the book’s flexible, modifiable, and modular structure, we believe we have written the first textbook for every instructor in the fields of learning and/or memory, reflecting and respecting the heterogeneity and diversity of the many different approaches to teaching this material.

Can this book be used for a Principles of Learning and Behavior course? Indeed it can.

Although more and more colleges are offering courses that integrate animal learning and human memory and include ever-increasing amounts of neuroscience, there are still numerous teachers who prefer to focus primarily on animal learning and conditioning, along with modest coverage of related studies of human associative learning, all presented primarily from a behavioral perspective.

For such a course, we recommend starting with **Chapter 1**, which lays a general foundation, then covering the four chapters of the Learning Module (**Chapters 3 through 6**) and concluding with

[Chapter 10](#), which examines key topics in fear conditioning. Together, these six chapters present a lucid, compelling, accessible, and engaging introduction to the principles of learning and behavior. We recognize, of course, that six chapters cannot provide as much detailed coverage as a single-approach textbook with 12 or more chapters on these topics. For this reason, we have included extensive additional materials on learning and behavior in the Instructor's Resource Manual. These materials provide the additional flexibility and content to support spending two weeks, rather than one week, on each of the four Learning Module chapters. This combination of textbook and supplemental materials serves well the teacher who wishes to spend 10 or more weeks on principles of learning and behavior with a primary focus on animal learning and conditioning.

Specialized learning and behavior textbooks are often dry and unappealing to most students. By adopting our book, instructors who prefer the learning and behavior approach will be providing their students with a text that has a uniquely engaging writing style, helpful integrated pedagogy, extensive four-color art, and a strong focus on showing students how basic research has direct implications for everyday life and clinical practice.

Neuroscience Focus

Neuroscience has altered the landscape for behavioral research, shifting priorities and changing our ideas about the brain mechanisms of behavior. *Learning and Memory: From Brain to Behavior*, Fourth Edition, integrates neuroscience research into each chapter, emphasizing how new findings from neuroscience have allowed psychologists to consider the functional and physiological mechanisms that underlie the behavioral processes of learning and memory. [Chapter 2](#) offers an accessible introduction to neuroscience for students unfamiliar with the basics of brain structure and function. Thereafter, the “Brain Substrates” section of each of the book's core chapters ([3](#) through [12](#)) presents the neuroscience perspectives relevant to the chapter topic, to be assigned or not as the teacher wishes (and omitted by those teachers who prefer to present only a behavioral perspective).

Integrated Presentation of Research Across Species

As scientists have gained a greater understanding of the neurobiological bases of behavior, the strict conceptual boundary that used to exist between the biological approach and the psychological approach to the study of learning and memory has largely disappeared. Moreover,

after several decades during which learning by humans was considered to be one field of science and learning by animals was considered to be another, the discovery of basic biological mechanisms common to all species has launched a unified approach to behavioral studies. Although our book focuses primarily on “learning” in one modular set of chapters and “memory” in another module, the story that emerges from both modules is, we believe, the strongest and most up-to-date representation available of the field as a whole.

Clinical Perspectives

In addition to examining and explaining new research in learning and memory, *Learning and Memory: From Brain to Behavior*, Fourth Edition, traces the development of new diagnoses and treatments for a variety of neurological and psychiatric disorders. Recent advances in neuroscience have produced dramatic changes in clinical practice over the past decade, greatly affecting how neurologists, psychiatrists, clinical psychologists, nurses, and rehabilitation specialists diagnose and treat a range of clinical disorders. Alzheimer’s disease, autism spectrum disorders, schizophrenia, Parkinson’s disease, dyslexia, anxiety disorders, ADHD, and stroke are just a sampling of the disorders for which new treatment options have been developed as a result of basic behavioral and cognitive neuroscience studies of learning and memory.

To reflect this broad impact of the field of learning and memory, each of the core chapters ([Chapters 3](#) through [12](#)) includes a “Clinical Perspectives” section that shows how knowledge of behavioral processes and brain substrates is being applied to understand clinical disorders that lead to disruptions of learning and memory. These sections are one way in which the book emphasizes the influence of learning and memory research in the real world and shows how neuropsychological research informs our understanding of memory mechanisms.

Student Friendliness

- **No Prerequisites.** We understand that students may come to this course from different backgrounds, even different disciplines, so we do not assume any previous level of familiarity with basic psychology or neuroscience concepts. The first two chapters of the text offer a comprehensive overview of the field of the psychology of learning and memory and the neuroscience foundations of behavior. Later chapters explain all new concepts clearly, with emphasis on real-life examples and with teaching-oriented illustrations.
- **Engaging Narrative.** Our aim has been to create a lively, clear, and example-rich narrative,

a colorful conversation between authors and readers that communicates our vision of an exciting field in transition and captures the interest of students by igniting their curiosity.

- **Full-Color Art Program.** The full-color art program consists of original anatomical art, state-of-the-art brain scans, color-coded graphs, and other vivid figures to help students visualize the processes involved in learning and memory. Photos offer a link to the real world, as well as a look back in time; cartoons provide occasional comical commentary (and often additional insights) alongside the main narrative.
- **Real-World Implications.** *Learning and Memory: From Brain to Behavior* is noted for a strong focus on applications and on the relevance of learning and memory concepts to everyday life. In addition to the “Clinical Perspectives” section at the end of every core chapter, we have included throughout each chapter many concrete, real-world examples of learning and memory that help students grasp the implications of what they are studying and its relevance in their own lives.
- **Consistent Organization.** The integration of both neuroscience and relevant clinical issues throughout the text is made more accessible to the student by the book’s consistent tripartite division of each chapter into the sections “Behavioral Processes,” “Brain Substrates,” and “Clinical Perspectives.” As described above, this also allows teachers to selectively omit the discussions of brain substrates or clinical perspectives from some or all of the reading assignments if that better suits a teacher’s syllabus. In addition, each chapter opens with a vignette that presents the chapter topic on a personal level, often with the story of a real individual (or, in one case, an accomplished dolphin named Akeakamai). The chapter then ends with a “Synthesis” discussion that recaps and integrates selected key issues in the chapter.

Extensive Pedagogy

Certain pedagogical aids are found throughout the body of each chapter. Others appear consistently at each chapter’s start or finish. Additional learning aids are part of the extensive supplements package, including a complete, ready-to-use package of PowerPoint slides for each section of every chapter.

- **Learning and Memory in Everyday Life** boxes in each chapter illustrate the practical implications of research, especially those that are relevant and interesting to undergraduate students. (Examples include “Unconscious Racial Bias,” “Extinguishing a Drug Habit,” and “How Much Stress Is Too Much?”)
- **Test Your Knowledge** exercises introduced at intervals throughout each chapter give

students the opportunity to check their comprehension and retention of more challenging topics immediately after having read about them. Suggested answers are provided at the end of the chapter. (Examples include “Maximizing Habituation,” “Reinforcement Versus Punishment,” and “Models of Working Memory.”)

- **Interim Summaries** follow each of the chapter’s principal sections to help students review major concepts presented in the pages they have just finished reading.
- **Quiz Yourself** fill-in-the-blank exercises at the end of each chapter test recall of key topics and concepts. Page references for where the information was presented are provided with each exercise, and answers are given in the Instructor’s Resource Manual.
- **Concept Checks** at the end of each chapter ask critical-thinking questions that require an understanding and synthesis of the key material in the chapter. These exercises ask students to apply the knowledge they’ve gained to a real-life situation. Suggested answers are provided in the Instructor’s Resource Manual.
- **Key Terms** are defined in the text margins for emphasis and easy reference and are also listed at the end of each chapter, with page references, to help students review chapter terminology. All key terms with their definitions are also included in an end-of-text glossary.

Media and Supplements

All of the supplementary materials can be downloaded from the Macmillan Learning catalog site, at www.macmillanlearning.com.

Book-Specific Lecture and Art PowerPoint Slides

To ease your transition to the fourth edition of *Learning and Memory*, a prepared set of lecture and art slides, in easily adapted and customizable PowerPoint format, is available to download from the catalog site. The book-specific PowerPoint Lecture Slides focus on themes and key terms within the text and include select illustrations, photos, graphs, and tables. The Image Slides include all illustrations, photos, graphs, and tables within the text. All slides can be used as they are or customized to suit your course needs.

Instructor's Resource Manual

The Instructor's Resource Manual includes extensive chapter-by-chapter suggestions for in-class presentations, projects, and assignments, as well as tips for integrating multimedia into your course. It provides answers to the Quiz Yourself items from each chapter and sample responses to the more open-ended Concept Check items. It also presents more comprehensive material on animal learning for instructors who allocate more of their courses to the classic studies of animal learning.

Test Bank

The Test Bank features approximately 100 questions per chapter, as well as an assortment of short-answer and essay questions. Each question is keyed to the American Psychological Association learning outcome for undergraduate psychology majors and Bloom's taxonomy. Visit macmillanlearning.com to access the test bank.

Acknowledgments: To Our Colleagues

This book has benefited from the wisdom of expert reviewers and instructors from laboratories and classrooms around the country. From the earliest stages of the development process, we solicited feedback and advice from the leading voices in the field of learning and memory to ensure that the book presents current and accurate understanding of the topics in each chapter. Over the course of the book's development, we have relied on these experts' criticism, corrections, encouragement, and thoughtful contributions. We thank them for lending us their insight, giving us their time, and above all for sharing in our commitment to creating a new textbook and a new curriculum that reflect a contemporary perspective on the field.

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Introductory Module

Chapter 1 Fundamental Themes in the Psychology of Learning and Memory



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Linking Ideas in the Mind

[William James and Memory Networks](#)

[*Learning and Memory in Everyday Life: Top 10 Tips for a Better Memory*](#)

[Ivan Pavlov's Conditioning Studies](#)

[Edward Thorndike and the Law of Effect](#)

Nature Versus Nurture

[Descartes and Dualism](#)

[John Locke and the Blank Slate](#)

[John Watson's Behaviorism](#)

[B. F. Skinner's Radical Behaviorism](#)

Comparing Humans to Other Animals

[Charles Darwin and the Theory of Natural Selection](#)

[The Neo-Behaviorism of Edward Tolman](#)

Formal Models and Universal Laws

[Hermann Ebbinghaus and Human Memory Experiments](#)

[*Learning and Memory in Everyday Life: What If You Could Form No New Memories?*](#)

[Clark Hull and Mathematical Models of Learning](#)

[W. K. Estes and Mathematical Psychology](#)

[George Miller and Information Theory](#)

[The Connectionist Models of David Rumelhart](#)

LIKE MANY OTHER WEALTHY YOUNG GREEKS OF HIS DAY, Aristotle (384–322 BC) was educated in Athens, the preeminent intellectual center of the western world at that time. There he studied under Plato (c. 427–347 BC), perhaps the greatest of the Greek philosophers, and became a philosopher and teacher of such stature himself that we call him “the father of Western thought.” His students included the young prince later known as Alexander the Great, who went on to conquer much of Europe and the Mediterranean.



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Aristotle (right) and his teacher, Plato

A keen observer of the natural world, Aristotle loved **data**, the facts and figures from which he could infer conclusions. He collected plants and animals from around the world and made careful notes about their structure and behavior. From such data, Aristotle attempted to formulate **theories**, sets of statements devised to explain a collection of facts. His data-oriented approach to understanding the world stood in marked contrast to the methods of his intellectual forebears, including Plato and Plato's teacher, Socrates, both of whom relied primarily on intuition and logic rather than natural observation.

data

Facts and figures from which conclusions can be inferred.

theory

A set of statements devised to explain a group of facts.

One of Aristotle's key interests was memory. His theory about it, called **associationism**, argued that memory depends on the formation of linkages ("associations") between pairs of events, sensations, or ideas, so that recalling or experiencing one member of the pair elicits a memory or anticipation of the other. Imagine someone reading a list of words to you and asking you to respond to each with the first word that comes to your mind. If he says "hot," you might say "cold"; if he says "chair," you might say "table," and so on. The words "hot" and "cold" are linked, or associated, in most people's minds, as are "table" and "chair." Aristotle formulated ideas about how these associations, these links between ideas in the mind, come about.

associationism

The principle that memory depends on the formation of linkages ("associations") between pairs of events, sensations, and ideas, such that recalling or experiencing one member of the pair elicits a memory or anticipation of the other.

Aristotle described such linkages as reflecting three fundamental principles, or universal laws, of association. The first principle is **contiguity**, or nearness in time or space: events experienced at the same time (temporal contiguity) or place (spatial contiguity) tend to be associated. The ideas of "chair" and "table" are linked because we often see chairs and tables together at the same times and in the same places. The second principle is *frequency*: the more often we experience events that are contiguous, the more strongly we associate them. Thus, the more often we see tables and chairs together, the stronger the table–chair link grows.

contiguity

Nearness in time (temporal contiguity) or space (spatial contiguity).

Aristotle's third principle is *similarity*: if two things are similar, the thought or sensation of one will tend to trigger a thought of the other. Chairs and tables are similar in that both are often made of wood, both are found in kitchens, and both have a function associated with eating meals. This similarity strengthens the association between them. Together, Aristotle concluded, these three principles of association—contiguity, frequency, and similarity—are universal principles through which humans organize sensations and ideas.

On the basis of these principles of learning, Aristotle argued that knowledge emerges from experience. This perspective identifies him with a philosophical school of thought known as **empiricism**, which holds that all the ideas we have are the result of experience (the Greek word

empiricus means “experience”). To Aristotle, the mind of a newborn child was like a blank slate, not yet written upon. On this issue, Aristotle differed sharply from his teacher Plato, who believed staunchly in [nativism](#), which holds that the bulk of our knowledge is inborn (or native). Plato’s most influential book, *The Republic*, described an idealized society in which people’s innate differences in skills, abilities, and talents form the basis for their fixed roles in life: some rule while others serve. The differing views of Aristotle and his teacher Plato, and the tension between empiricism and nativism, have continued through the centuries to fuel scientific discussion, although today the topic is more often called the “nature versus nurture” debate: researchers argue about whether our “nature,” including genes, or our “nurture,” including upbringing and environment, has the greater influence on our learning and memory abilities.

empiricism

A philosophical school of thought that holds that all the ideas we have are the result of experience.

nativism

A philosophical school of thought that holds that the bulk of knowledge is inborn (or native).

But what about other animals, like dogs, sheep, and cows? Do they learn the same way humans do? Can they form and recall memories? Aristotle believed that humans exist on the top of a hierarchical continuum of animals, with both similarities and differences between us and other creatures. In his treatise on *The History of Animals*, Aristotle wrote, “Many animals have memory, and are capable of instruction; but no other creature except man can recall the past at will.” Do you agree with this? Later chapters describe research showing that while all animals, including humans, do have memory, humans have some very advanced capabilities for controlling and managing what aspects of memory are to be recalled.

This book is about [learning](#), the process by which changes in behavior arise as a result of an organism’s experience interacting with the world, and about [memory](#), the organism’s internal record of past experiences, acquired through learning. Much of what we know about learning and memory builds on the contributions of Aristotle. [Chapter 2](#), for example, introduces the influences of contiguity and frequency on the neural bases of learning; [Chapters 4](#) and [5](#) describe modern behavioral and neurobiological studies of the interaction between contiguity and frequency in learning; [Chapter 6](#) shows why similarity continues to be a core focus of research on learning; and [Chapter 12](#) dwells extensively on issues of genetics and environment. The questions that are examined today in research on learning and memory conducted in laboratories around the world are simply extensions of Aristotle’s early struggles to understand how learning

and memory work.

learning

The process by which changes in behavior arise as a result of experiences interacting with the world.

memory

The internal record of past experiences acquired through learning.

Four essential themes—fundamental questions and challenges—emerge from Aristotle’s thinking and writing about learning and memory. We can summarize them as follows:

1. How do sensations or ideas become linked in the mind?
2. To what extent are behaviors and abilities determined by biological inheritance (nature) and to what extent by life experiences (nurture)?
3. In what ways are human learning and memory similar to learning and memory in other animals, and in what ways do they differ?
4. Can the psychological study of the mind be rigorously scientific, uncovering universal principles of learning and memory?

The rest of this chapter is organized around historical progress that philosophers and psychologists have made in answering these four questions. While reading about these key contributions and themes, you will also be introduced to essential concepts and terms that appear throughout the book, and you will begin to see why the questions that fascinated Aristotle are as relevant today as ever.

1.1 Linking Ideas in the Mind

How do sensations or ideas become linked in the mind? What determines when and if we will form an association between two ideas, link two stimuli or events in our mind, or come to associate a given behavior with a desirable (or undesirable) outcome? Building on the philosophical ideas developed by Aristotle over 2,000 years ago, several early psychologists led the search for concrete answers to these questions.

William James and Memory Networks

Born to a wealthy and prominent New York family, William James (1842–1910) spent his early years traveling around the world, living in fine hotels, and meeting many of the great writers and philosophers of his time. After receiving his medical degree in 1869, James accepted a position as an instructor of physiology and anatomy at Harvard, where he offered an introductory course in [psychology](#), the study of mind and behavior. It was the first course on psychology ever given at any college in America. James often joked that the first psychology lecture he ever attended was his own.



(MS Am 1092.) Houghton Library, Harvard University

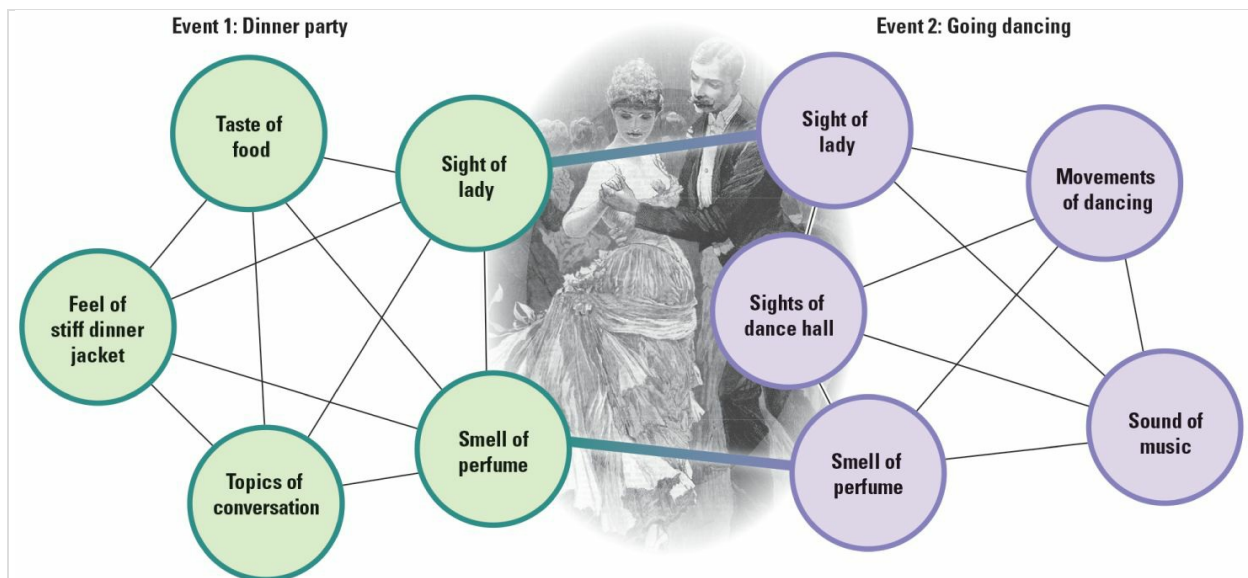
William James

psychology

James's introductory psychology course soon became one of the most popular courses at Harvard, and he signed a contract with a publisher, promising to deliver, within 2 years, a book based on his acclaimed lectures. In the end, it took him 12 years to finish the book, but James's two-volume [*Principles of Psychology* \(1890\)](#) was an immediate scientific, commercial, and popular success. Translated into many languages, it was for decades the standard psychology text around the world.

James was especially interested in how we learn new habits and acquire new memories. He enjoyed telling the story of a practical joker who, seeing a recently discharged army veteran walking down the street carrying a load of groceries, shouted, "Attention!" The former soldier instantly and instinctively brought his hands to his side and stood ramrod straight as his mutton and potatoes rolled into the gutter. The soldier's response to this command was so deeply ingrained, even after he had left the army, that it was all but impossible to suppress. Like Aristotle, James believed that most abilities and habits are formed by our experiences, especially early in life. James proposed that a central goal of psychology should be to understand the principles that govern the formation and maintenance of new skills and memories, including how and why old learning may block or facilitate the formation of new learning ([James, 1890](#)); indeed, this tension between old memories and new learning has been an ongoing focus of experimental psychology in the past century, as reviewed in many other chapters in this book, especially [Chapters 7](#) and [8](#).

James was a strong proponent of associationism, and his writings elaborated on the work of Aristotle. The act of remembering an event, such as a dinner party, he wrote, would involve a network of multiple connections between the components of the evening. These might include memories for the taste of the food, the feel of his stiff dinner jacket, and the smell of the perfume of the lady seated next to him ([Figure 1.1](#)). Activation of the memory network for the dinner party, with all of its component associations, could in turn activate the memory of a second event that shared some related elements—such as a visit to a dance hall with the same lady on the next night. This second event would be composed of its own parts: the sights of the dance hall, the movements of dancing, the smell of his partner's perfume, and so on. The two events (dinner party and dancing) would be associated by a linkage between their common or related components (the sight of the lady and the smell of her perfume).



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers
Image from Mansell/Time Life Pictures/Getty Images

FIGURE 1.1 William James’s memory model Memory of an event, such as a dinner party, has multiple components all linked together. Another event, such as going dancing with a lady from the dinner party, also has component parts linked together. A mental association between the two events in turn consists of multiple connections between components common to both.

Description

The flowchart consists of two parts. They are Event 1, Dinner party, and Event 2, Going dancing. The flowchart for Dinner party event has five interconnected circles, Taste of food, Sight of lady, Smell of perfume, Topics of conversation, and Feel of stiff dinner jacket. The flowchart for Going dancing event also has five interconnected circles, Sight of lady, Movements of dancing, Sights of dance hall, Smell of perfume, and Sound of music.

The sight of lady from Event 1 is connected to the Sight of lady from Event 2. The smell of perfume from Event 1 is connected to the Smell of perfume from Event 2.

A photo on the right shows a young man, in a tailored suit, dancing with a woman wearing a decorated flared dress. The man has held the woman by her hand. The background shows a large group of couples dancing.

This *model*, or simplified description, of memory networks was one of James’s many seminal contributions to psychology. Today, most modern theories of memory draw on this idea of learning as a process of forming associations between the elements of an experience—as demonstrated in [Chapters 4](#) and [7](#), among others. (For advice on forming memory networks to improve your own memories, see “[Learning and Memory in Everyday Life](#)”.) James himself took the model literally, believing that the network of associations it described would eventually be mapped directly onto physical connections in the brain ([James, 1890](#)). Such an expectation

was far ahead of its time: linking physical brain processes to learned behaviors would not attract much interest for many decades.

LEARNING AND MEMORY IN EVERYDAY LIFE

Top 10 Tips for a Better Memory

1. *Pay attention.* Often when we “forget” something, it is not that we have somehow lost the memory of it but that we did not learn the thing properly in the first place. If you pay full attention to what you are trying to learn, you will be more likely to remember it later. (This principle is discussed further in [Chapter 7](#).)
2. *Create associations.* Associate what you are trying to learn with other information you already know. For example, it will be easier to remember that Ag is the chemical symbol for silver if you know that it is short for *argentum*, the Latin word for “silver.” It might also help if you know that Argentina got its name from early European explorers who mistakenly thought the region was rich in silver. (This principle is discussed further in [Chapter 7](#).)
3. *A picture is worth a thousand words.* Names and dates and such are more memorable if you can link them to an image. The effort you expend generating an image strengthens the memory. For example, in an art history course, you might have to remember that Manet specialized in painting human figures, whereas his contemporary Monet is famous for paintings of haystacks and water lilies. Picture the human figures lined up acrobat-style to form a letter “A” for Manet and the water lilies arranged in a daisy chain to form the letter “O” for Monet. (This principle is discussed further in [Chapter 7](#).)
4. *Practice makes perfect.* There is a reason to drill kindergarteners on their ABCs and make third-graders repeatedly recite their multiplication tables. Memories for facts are strengthened by repetition. The same principle holds for memories for skills, such as bike riding and juggling: they are improved by practice. (This principle is discussed further in [Chapter 8](#).)
5. *Use multiple senses.* Instead of just reading information silently, read it aloud. You will encode the information aurally as well as visually. You can also try writing it out; the act of writing activates sensory systems and also forces you to think about the words you are copying.
6. *Reduce overload.* Using memory aids such as sticky notes, calendars, or electronic schedulers to remember appointments, due dates, and other obligations frees you to focus on remembering items that must be called to mind without written aids—say, during an exam! (This principle is discussed further in [Chapter 9](#).)
7. *Time travel.* Remembering facts does not depend on remembering the exact time and place where you encountered them. Nevertheless, if you can’t remember a fact, try to remember where you first heard it. If you can remember your high school history teacher lecturing on Napoleon, perhaps what she said about the causes of the Napoleonic Wars will also come to mind. (This principle is discussed further in [Chapter 7](#).)
8. *Get some sleep.* Two-thirds of Americans do not get enough sleep. Consequently, they are less able to concentrate during the day, which makes it harder for them to encode new memories and retrieve old ones (see Tip 1). Sleep is also important for helping the brain organize and store memories. (This principle is discussed further in [Chapter 7](#).)
9. *Try a rhyme.* Do you have to remember a long string of random information? Create a poem (or, better yet, a song) that includes the information. Remember the old standard “I’ before ‘E’ except after ‘C’ or sounded as ‘A,’ as in ‘neighbor’ or ‘weigh’”? This ditty uses rhythm and rhyme to make it easier to remember a rule of English spelling.
10. *Relax.* Sometimes trying hard to remember is less effective than turning your attention to something else; often, the missing information will pop into your awareness later. If you are stumped by a question on a test, skip that one and come back to it later, when perhaps the missing information will not be so hard to retrieve. (This principle is discussed further in [Chapter 7](#).)

Ivan Pavlov's Conditioning Studies

The Russian physiologist Ivan Pavlov (1849–1936) is known for developing methods for studying animal learning that are still in widespread use today. As a young man, Pavlov trained to be a Russian Orthodox priest, like his father and grandfather had been. In addition to his religious readings, he read Charles Darwin's recently published *The Origin of Species*. Inspired by Darwin's accomplishments in biology and natural history (discussed in [Section 1.3](#)), Pavlov abandoned his plan of becoming a priest and enrolled in the school of natural sciences at the University of St. Petersburg.



RIA Novosti/Science Source

Ivan Pavlov

Although remembered today for his seminal contributions to the psychology of learning, Pavlov's 1904 Nobel Prize in Physiology or Medicine was actually awarded for his research on the physiology of salivation and digestion in dogs (see [Figure 1.2A](#)). His landmark studies of the factors that influence how an animal learns (described in detail in [Chapter 4](#)) consisted of

inserting surgical tubes into the mouths of dogs to collect saliva and measure the amount of salivation in response to various cues. In one study, he began by training a dog to expect that the sound of a doorbell always preceded delivery of food; over many trials in which the sound of the doorbell was paired with food, the dog developed a stronger and stronger salivation response to the sound of the bell. In this form of learning, an animal learns, to the extent of responding reflexively, that a certain **stimulus**, or sensory event (in this case, a doorbell), predicts the occurrence of a second, important event (in this case, delivery of food). Known today as **classical conditioning** (or Pavlovian conditioning), it is a fundamental mechanism by which two concepts, such as a bell and food, come to be linked or associated in our minds.

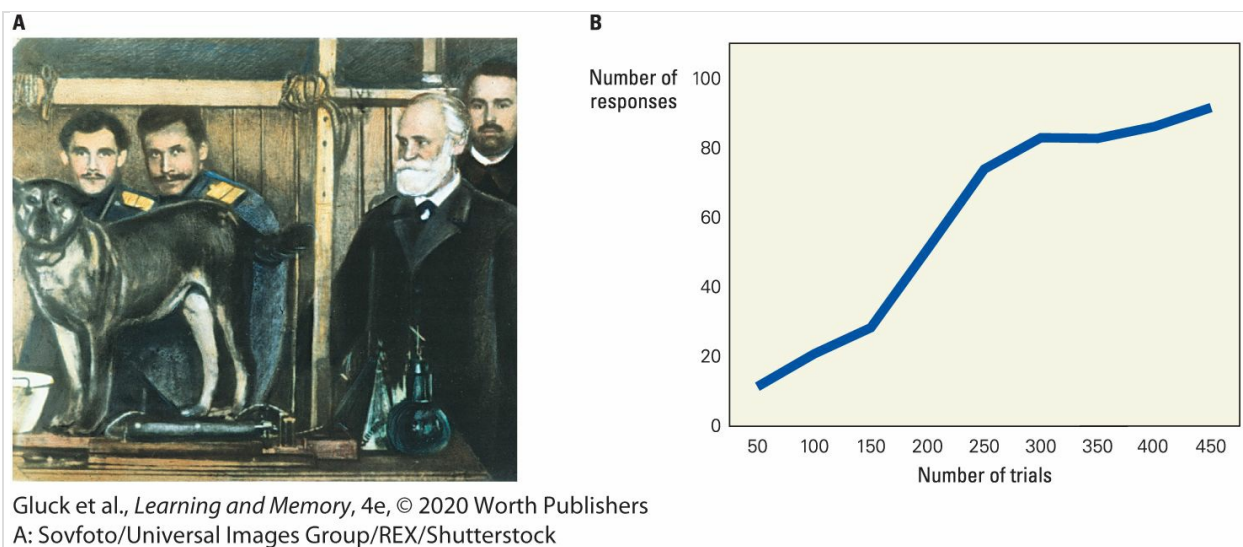


FIGURE 1.2 Pavlov and learning experiments (A) Pavlov (with white beard) and his assistants in the laboratory, along with one of the dogs they used in their studies of the salivary response. (B) A learning curve from a modern study of classical conditioning. The curve plots the number of training trials (the independent variable) against the animal's conditioned response (the dependent variable): as the training trials accumulate, the animal's responses increase, showing that the learning starts off slowly, gets faster in the middle of the process, and later tapers off and again slows down.

Description

The photo on the left shows Ivan Pavlov with his three assistants in a laboratory. They are posing for a picture along with a dog used in one of their experiments. The graph on the right plots a curve of classical conditioning. The horizontal axis represents the number of trials. The vertical axis represents the number of responses. The curve begins at point (50, 10) and gradually increases to (450, 90).

stimulus

A sensory event that provides information about the outside world.

classical conditioning

A type of learning in which the organism learns to respond to a previously neutral stimulus that has been repeatedly presented

along with a biologically significant stimulus; also called *Pavlovian conditioning*.

Modern studies of classical conditioning usually report the results as a **learning curve**, like that shown in [Figure 1.2B](#). On the horizontal axis of [Figure 1.2B](#) is plotted the number of training trials. This value is the **independent variable**, the factor being manipulated in the experiment. Against the number of training trials is plotted, on the vertical axis, the strength or frequency of the animal's **response**, the behavioral reaction the stimulus evokes. This value is the **dependent variable**, the factor under observation. The learning curve shows how changes in the independent variable affect the dependent variable.

learning curve

A graph showing learning performance (the dependent variable, usually plotted along the vertical axis) as a function of training time (the independent variable, usually plotted along the horizontal axis).

independent variable

The factor that is manipulated in an experiment.

response

The behavioral consequence of perception of a stimulus.

dependent variable

In an experiment, the factor whose change is measured as an effect of changes in the independent variable.

Pavlov's view of how an animal learns a new association was based on an analogy to a new technology that had recently been introduced in Russia: the telephone. As Pavlov explained it, he could call his lab from home via a *direct* private line, which was a fixed connection, much like the fixed connection between food and salivation in a dog's brain. Alternatively, he could call his lab by going through an *indirect* pathway, via a switchboard operator, creating a modifiable connection, much like the newly learned connection between the bell and the dog's salivation ([Pavlov, 1927](#)).

In other studies, Pavlov and his assistants showed that they could also *weaken* an animal's trained response to the bell. This was done by first pairing the bell with food, until the animal had learned to salivate to the bell, and then pairing the bell with the *absence* of food. Pavlov called this process **extinction**: the salivation to the bell gradually decreased as the animal learned that the bell no longer predicted food.

extinction

The process of reducing a learned response to a stimulus by ceasing to pair that stimulus with a reward or punishment.

Pavlov also demonstrated that a dog will transfer what it has learned about one stimulus to

similar stimuli. For example, he observed that once a dog learned to respond to a metronome ticking at 90 beats per minute, it also responded to similar sounds, such as a metronome ticking at 80 beats per minute or 100 beats per minute. However, the more the new stimulus differed from the original stimulus, the less the dog salivated. These graded responses to stimuli of varying dissimilarity to the original training stimulus are an example of **generalization**, the transfer of past learning to novel events and problems. [Chapter 6](#) discusses how generalization occurs in many different forms of learning and memory.

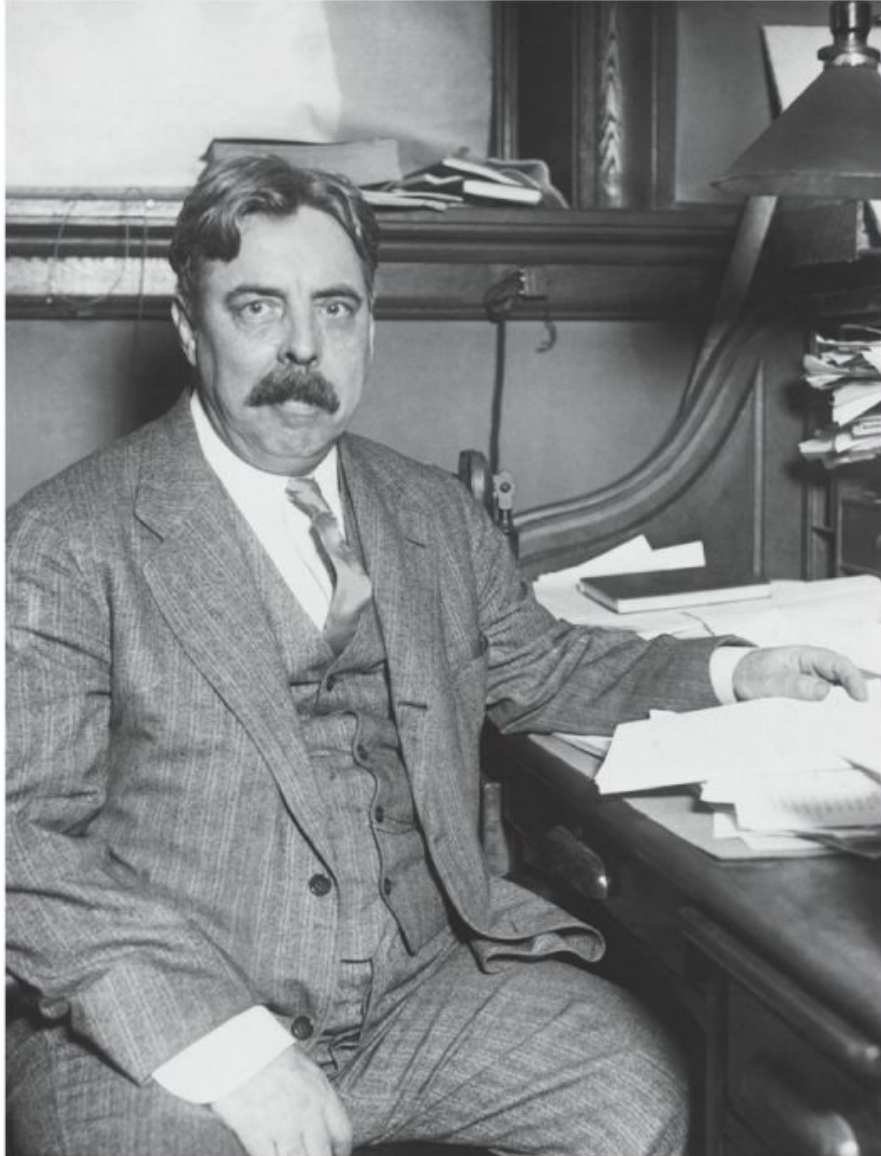
generalization

The transfer of past learning to novel events and problems.

Ivan Pavlov lived through the Russian Revolution of 1917 and developed a deep animosity toward the new Communist regime (especially after they confiscated his Nobel Prize money). Nevertheless, when Pavlov died, in 1936, he was given an elaborate funeral with full honors as a hero of the Soviet state.

Edward Thorndike and the Law of Effect

Back in the United States, Edward Thorndike (1874–1949), a student of William James, was also studying how animals learn associations—in this case, the connection between a stimulus and a behavioral response that leads to a desirable outcome. Some of Thorndike’s most influential studies involved observing how cats learn to escape from puzzle boxes—cages secured with complex locking (and unlocking) devices. Thorndike called this kind of training, in which organisms learn to make responses in order to obtain or avoid important consequences, *instrumental conditioning* because the organism’s behavior is instrumental in determining whether the consequences occur. This is in contrast, for example, to the learned response (salivation) of Pavlov’s dogs, in which the dogs received their food reward regardless of whether they made the learned response. Instrumental conditioning, now more commonly referred to as **operant conditioning**, is the focus of [Chapter 5](#).



Humanities and Social Sciences Library/
New York Public Library/Science Photo
Library/Science Source

Edward Thorndike

operant conditioning

The process whereby organisms learn to make responses in order to obtain or avoid important consequences; also called *instrumental conditioning*.

In his studies, Thorndike observed that the probability of a particular behavioral response

increased or decreased depending on the consequences that followed. He called this the **law of effect** ([Thorndike, 1911](#)). If a particular response led to a desirable consequence, such as access to food, then the probability of the animal making that response in the future *increased*. On the other hand, if the response led to an undesirable consequence (say, an electric shock), then the probability of the animal making that response in the future *decreased*. Fascinated, Thorndike began to methodically investigate the factors that influence how an animal learns new behaviors to maximize its chances of obtaining desirable consequences and avoiding undesirable ones.

law of effect

The observation, made by Thorndike, that the probability of a particular behavioral response increases or decreases depending on the consequences that have followed that response.

According to the law of effect, an animal has a range of behaviors: behaviors that lead to positive consequences for the animal tend to persist; those that do not tend to die out. Starting from this basic principle, Thorndike argued that the psychology of learning should center on the search for the rules describing how, when, and to what degree associations among stimuli and responses are increased or decreased through experience ([Thorndike, 1932](#), [1949](#)).

In 1917, Thorndike became the first psychologist elected to the prestigious U.S. National Academy of Sciences, and in the early 1920s, he was often identified as one of the most influential scientists in the United States.

Interim Summary

- William James studied how people learn habits and acquire new memories, positing that memories are networks of associations, with similar memories sharing key elements in common.
- Ivan Pavlov demonstrated how psychologists can study and measure learning about associations that exist in the world.
- Edward Thorndike showed that animals learn to associate given behaviors with desirable or undesirable outcomes.

1.2 Nature Versus Nurture

Recall the differing views of Aristotle and his teacher Plato about whether our “nature” or our “nurture,” including upbringing and environment, has the greater influence on our learning and memory abilities and who we are as people. Continuing this debate were four key contributors to the psychology of learning and memory, whom we will consider next. The nature–nurture debate continues today, informing our efforts to understand how and to what degree we are able to change and evolve within the span of our own lifetimes, a topic covered in more detail in [Chapter 12](#). Today, most researchers take the middle road, acknowledging the profound influence of genes (nature) on learning and memory while noting that a lifetime of experience (nurture) modifies these influences.

Descartes and Dualism

In contrast to Aristotle, who believed knowledge was attained through experience, René Descartes (1596–1650) had no interest in theories of learning. He acknowledged that people do derive some information from experience, but he believed that much of what we know is innate.

Descartes grew up in France, the son of a provincial noble family. His family inheritance gave him the freedom to spend his life studying, thinking, and writing—most of which he did in bed (he hated to get up before noon). Although raised as a Roman Catholic and trained by Jesuits, Descartes harbored deep doubts about the existence of everything. Despairing of being able to know anything for certain, he concluded that the only evidence that he himself even existed was his ability to think: “*Cogito ergo sum*,” or “I think, therefore I am” ([Descartes, 1644](#)).

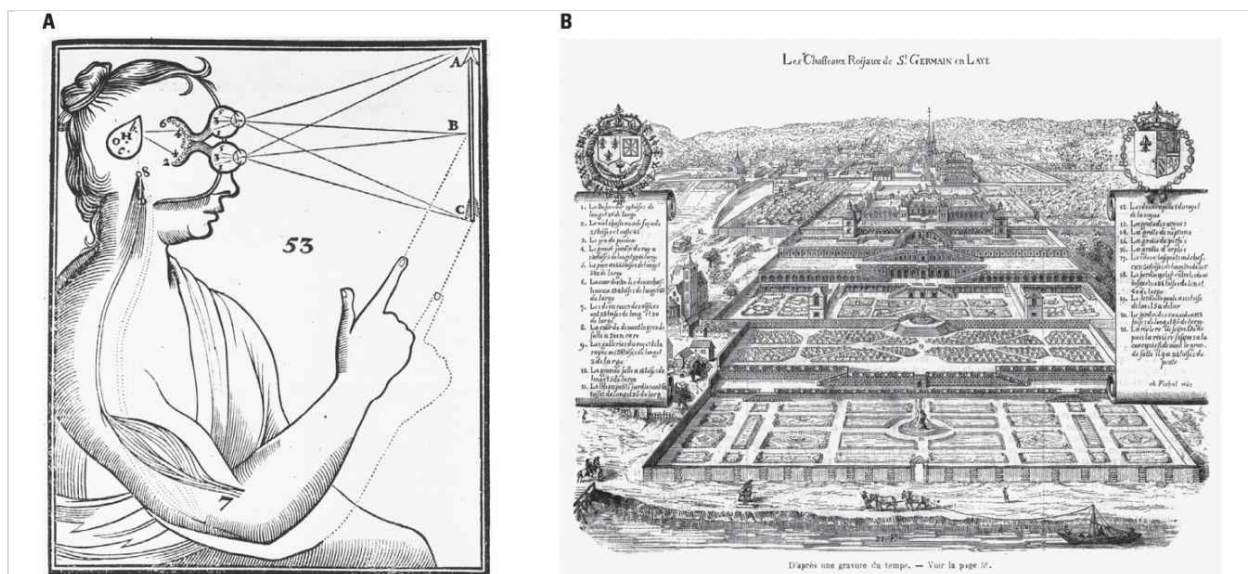


Portrait of René Descartes (1596–1650)
(oil on canvas)/French School, 17th
century/Musée des Augustins, Toulouse,
France/Bridgeman Images

René Descartes

Where does Descartes's *cogito*—the ability to think—come from? He was a firm believer in **dualism**, the principle that the mind and body exist as separate entities, each with different characteristics and governed by its own laws ([Descartes, 1662](#)). The body, Descartes reasoned,

functions like a self-regulating machine, much like the clockwork statues and fountains that were so fashionable during the Renaissance. A person strolling through the royal gardens of Saint-Germain-en-Laye, just outside Paris, would step on a hidden trigger, releasing water into pipes that caused a gargoyle to nod its head, a statue of the god Neptune to shake its trident, and the goddess Diana to modestly retreat. Descartes envisioned the body as working through a similar system of hydraulics and switches. The process begins when a stimulus from the outside world enters the system; for example, light reflected off a bird enters the eye as a visual stimulus. Like the trigger switch in the gardens, this stimulus causes fluids (Descartes called them “spirits”) to flow through hollow tubes from the eyes to the brain and then to be “reflected” back as an outgoing motor response, the behavioral consequence of perception of the stimulus, as illustrated by Descartes’s sketch in **Figure 1.3** (Descartes, 1662). Such a pathway from sensory stimulus to motor response is called a **reflex arc**.



A: Corbis Historical/Getty Images; B: Historia/Shutterstock

FIGURE 1.3 Descartes’s reflex (A) A mechanism for producing an automatic reaction in response to external events, as illustrated in Descartes’s *De Homine* (1662). The diagram shows the flow of information from the outside world, through the eyes, to the brain, and then through the muscles of the arm, creating a physical response in which the arm moves to point to an object in the external world. (B) The royal gardens of Saint-Germain-en-Laye, where Descartes found inspiration for his mechanistic approach to behavior.

Description

The illustration on the left shows the theory of vision from Rene Descartes *DE HOMINE*, Treatise on Man, 1677. A labeled human outline has a pair of eyes pictured above each other and focused on an arrow at the right. Lines extend from the eyes to the middle and terminal points of the arrow. The ends of the arrow are labeled A and C, and the middle

point is labeled B. Within the head, lines connect to the optic nerves of the gland and lead to the arm which points at the arrow.

The illustration on the right shows the royal gardens of Saint-Germain-en-Laye.

dualism

The principle that the mind and body exist as separate entities.

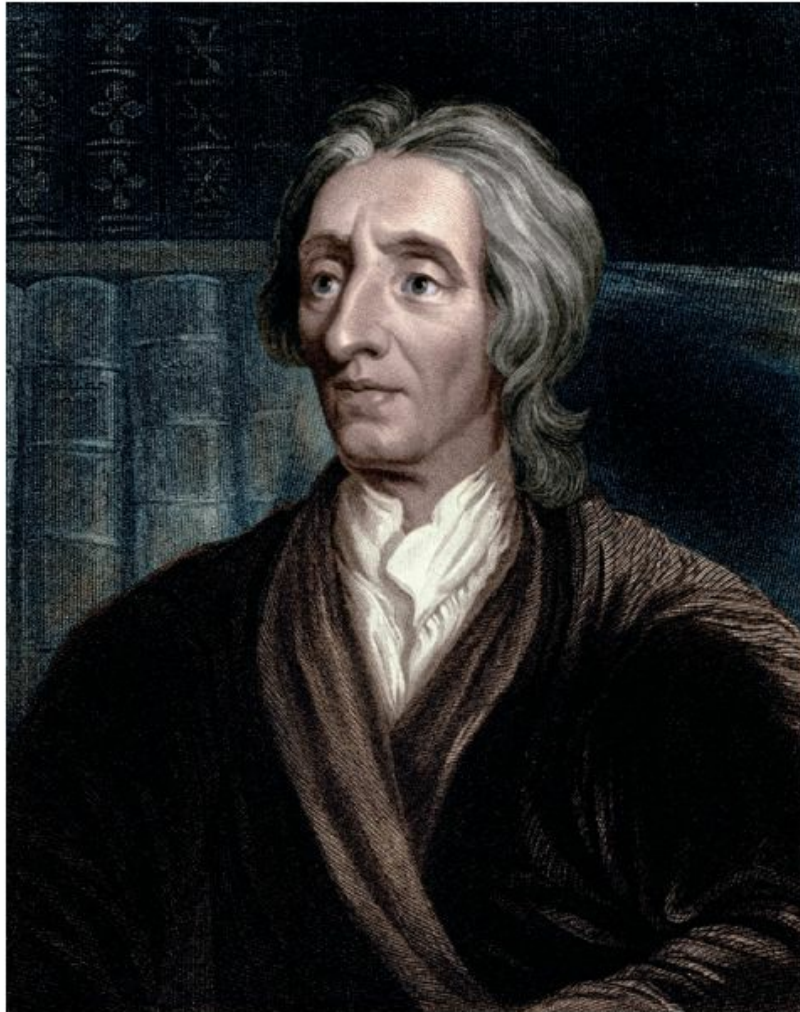
reflex arc

An automatic pathway from a sensory stimulus to a motor response.

Descartes got many of the details of reflexes wrong. There are no spirits that flow through the body to produce hydraulic movement as he described. Nevertheless, Descartes was the first to show how the body might be understood through the same mechanical principles that underlie physical machinery. This mechanistic view of the processes that give rise to behavior returned in full force many centuries later, in the mathematical and computer models of the brain and behavior that are described in several chapters in this book.

John Locke and the Blank Slate

In contrast to Descartes's idea of innate knowledge and built-in reflexes, John Locke (1632–1704) argued that all knowledge is derived from experience. Borrowing Aristotle's analogy of a tablet on which nothing is yet written, Locke suggested that children arrive in the world as a blank slate or tablet (in Latin, a *tabula rasa*) just waiting to be written on.



INTERFOTO/Alamy Stock Photo

John Locke

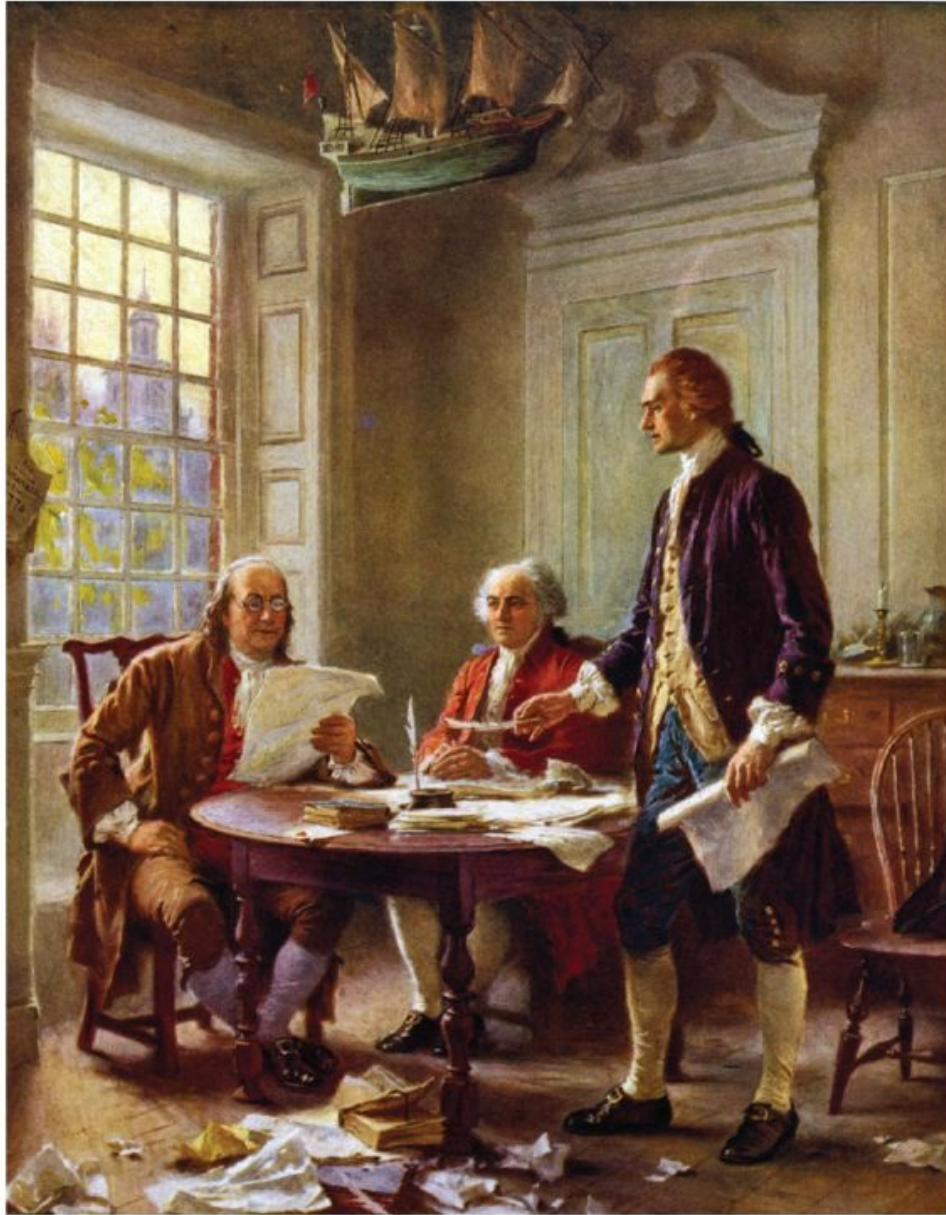
Locke did most of his work during the late 1600s in England, a time when science flourished. Famous scientists were the celebrities of their day; people attended lectures on philosophy and natural sciences the way they now go to movies and popular music concerts. One especially renowned scientist, Isaac Newton, demonstrated that white light can be refracted into component colors by a prism lens and then recombined by another lens to produce white light again.

Inspired by Newton's work, Locke hoped to show that the mind, too, could be broken down into elements that, when combined, produced the whole of consciousness. In this way, Locke borrowed methods from the physical sciences that he hoped would help him better understand

the mind and the processes of learning and memory. This practice of philosophers and psychologists of borrowing from other, more established and rigorous domains of science continues to this day.

To describe the way elementary associations might account for the more complex ideas and concepts that make up our memories and knowledge, Locke drew from the work of his former Oxford medical instructor, Robert Boyle, who 30 years earlier had demonstrated that chemical compounds are composed of elementary parts (what we now know to be molecules and atoms). Locke reasoned that complex ideas are similarly formed from the combination of more elementary ideas that we passively acquire through our senses ([Locke, 1690](#)). For example, simple ideas such as “red” and “sweet” are acquired automatically by our senses of sight and taste, and more complex ideas such as “cherry” are acquired by combining these simpler components.

Locke’s view of the power of experience to shape our capabilities through a lifetime of learning had great appeal to reformers of the eighteenth century, who were challenging the aristocratic system of government, in which kings ruled by right of birth. Locke’s ideas meant that a person’s worth was not determined at birth. All are born equal, he believed, with the same potential for knowledge, success, and leadership. Common people, through striving and learning, could transcend the limits and barriers of class. Therefore, Locke argued, access to a good education should be available to all children, regardless of their class or family wealth ([Locke, 1693](#)). These ideas heavily influenced Thomas Jefferson as he drafted the Declaration of Independence, which in 1776 proclaimed the American colonies’ independence from Great Britain and asserted that “all men are created equal,” with the same innate rights to “life, liberty, and the pursuit of happiness”—words taken almost verbatim from Locke’s writings.



Universal History Archive/ UIG/Shutterstock

Thomas Jefferson (on right) shares his draft of the Declaration of Independence with Benjamin Franklin and John Adams. If Thomas Jefferson had been inspired by Descartes, rather than Locke, how might the Declaration of Independence have been different?

Although Locke's writings were influential throughout European philosophical and scientific circles, he was not without critics. One of Locke's contemporaries, German mathematician Gottfried Wilhelm Leibniz, conceded to Locke that three-quarters of knowledge might be acquired but claimed that the other quarter is inborn and innate, including habits, predispositions,

and potentials for success or failure ([Leibniz, 1704](#)). In many ways, Leibniz's more nuanced position echoes the stand adopted by numerous modern researchers, who believe that human ability is not due solely to nature (nativism) or solely to nurture (empiricism) but is a combination of both.

John Watson's Behaviorism

In the 1920s, a new approach to learning, called [behaviorism](#), emerged. Behaviorism argued that psychology should restrict itself to the study of observable behaviors (such as pressing levers, salivating, and other measurable physical actions or activities) and avoid reference to unobservable, often ill-defined, internal mental events (such as consciousness, intent, memory, and thought). Proponents of this approach, who were called *behaviorists*, wanted to distance themselves from philosophers and psychologists who explored the inner workings of the mind through personal introspection and anecdotal observation.



George Rinhart/Corbis Historical/Getty Images

John Watson

behaviorism

A school of thought that says psychology should restrict itself to the study of observable behaviors (such as lever presses, salivation, and other measurable actions) and not seek to infer unobservable mental processes.

Brash, ambitious, and self-made, John Watson (1878–1958) is considered the founder of behaviorism. He was born in Greenville, South Carolina, to a deadbeat father who abandoned the family when Watson was 13 years old. Although a poor student as a child, Watson not only finished college but went on to graduate school, where he conducted research on how rats learn.

In these studies, Watson placed a rat at the entrance to a maze and rewarded it with food if it found its way through the corridors to the exit.

Initially, a “naive” (i.e., untrained) rat might spend half an hour wandering randomly through the maze until it reached the exit. After 30 training trials, however, the rat could traverse the maze in less than 10 seconds. To find out what drove the rat’s performance, Watson systematically eliminated various possibilities. First, he trained rats to run through the maze under normal conditions. Then he surgically blinded the rats, or rendered them deaf, or removed their whiskers (which rats use like fingertips to feel their way). None of these treatments impaired the rats’ performance. Thinking the rats might be using olfactory cues to find their way, Watson boiled the mazes to eliminate all odors. The rats still found their way through. Only when the maze was rotated or when the corridors were made shorter or longer did the rats show a significant loss in their ability to navigate the maze. From these studies, Watson argued that the rats had learned an automatic set of motor habits for moving through the maze and that these motor habits were largely independent of any external sensory cues—but could be disrupted by changing the lengths of the various arms, and thus the points at which the rats should make turns ([Watson, 1907](#)). Such learned motor habits, or skills, are discussed further in [Chapter 8](#).

According to Watson, psychology should be viewed as a “purely objective experimental branch of natural science. Its theoretical goal is the prediction and control of behavior” ([Watson, 1913](#)). Watson was a strong empiricist, sharing Locke’s belief in the overwhelming influence of experience (nurture) versus heredity (nature) in determining our behaviors and capabilities. In a rousing affirmation of Aristotle’s principle of the blank slate, Watson wrote:

Give me a dozen healthy infants, well-formed, and my own specified world to bring them up in, and I’ll guarantee to take any one at random and train him to become any type of specialist I might select—doctor, lawyer, artist, merchant, chief, and yes even beggarman and thief, regardless of the talents, penchants, tendencies, abilities, vocations, and race of his ancestors.

[\(Watson, 1924, p. 82\)](#)

In the years following World War I, many people hoped for a new dawn of equal opportunity and freedom from class-based constraints on social progress. Watson’s bold claims had a strong

appeal for scientists and the wider public.

Watson's career as an academic researcher came to a sudden end, however, when he became involved in a romantic relationship with his research assistant, Rosalie Rayner. Given Watson's fame as a scientist, his status as a married man, and Rayner's socially prominent family, the affair received intense media scrutiny. In the end, the scandal grew so great that Johns Hopkins University made Watson choose between ending his affair and resigning from his position at the university. Choosing to stay with the woman he loved, Watson resigned from Johns Hopkins and started a successful career in advertising, where he applied the same strict scientific principles to applied marketing research as he had to his earlier basic research on psychology. Much of modern advertising, like much of modern learning research, is rooted in the early seminal contributions of Watson.

B. F. Skinner's Radical Behaviorism

Watson may have been the father of behaviorism, but it was Burrhus Frederic Skinner (1904–1990), born in rural Pennsylvania, who became the most famous—and perhaps most infamous—behaviorist of the twentieth century. Although his original goal was to be a writer, Skinner instead went to graduate school in psychology. He placed himself squarely in the behaviorist camp, believing that psychologists should limit themselves to the study of observable behaviors that can be learned through experience, and not try to speculate about what is going on in the mind of an animal while it learns.



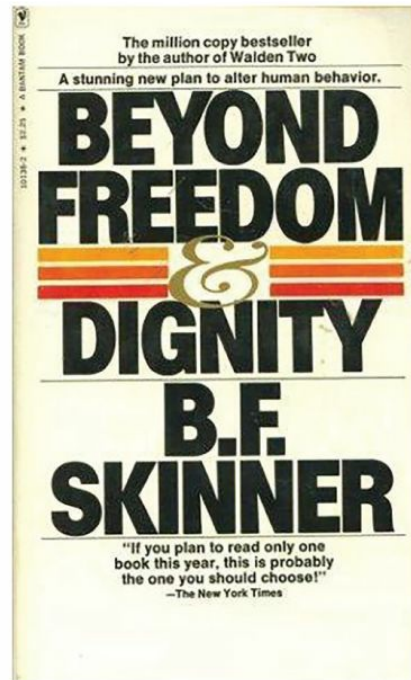
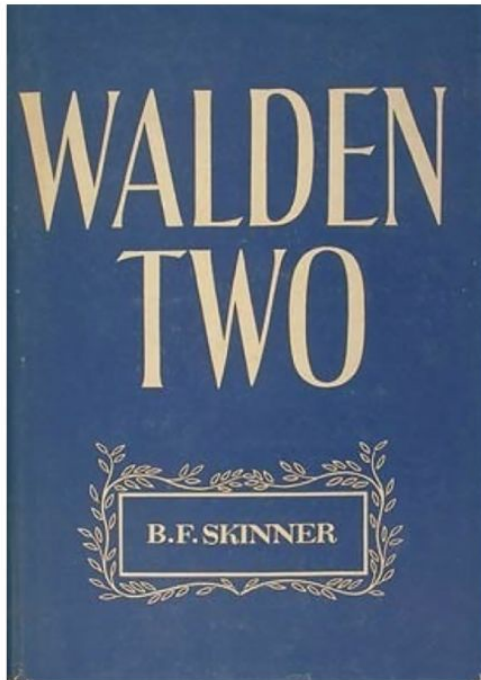
Bettmann/Getty Images

B. F. Skinner

Skinner extended and refined the techniques Thorndike had developed to study how animals learn new responses. For example, he built an automated learning apparatus that was widely adopted by others, who dubbed it the “Skinner box.” ([Chapter 5](#) includes more details about this and Skinner’s other innovations.) He also made many important contributions to our understanding of how animals learn associations between responses and consequences. One of his most important discoveries happened by accident.

In the early 1940s, Skinner was in his laboratory on a Friday afternoon, setting up some studies in which he taught rats to perform a response in order to obtain food pellets. He realized he did not have enough food pellets to get him through all the experiments planned for that weekend. Rather than cancel the experiments or go out and get more rat food, Skinner decided to save pellets by providing food only after the rats made two or three correct responses in a row. This led Skinner to one of his greatest discoveries: when trained with intermittent reinforcements, rats learn to respond as frequently as when they are rewarded on every trial—in fact, sometimes even more frequently. Skinner and his students began a massive new program of research on how learning is affected by the reliability with which an organism's responses result in consequences (such as obtaining a food pellet).

Today, B. F. Skinner's name is far better known than Watson's or Thorndike's because his influence extended beyond the laboratory. Fulfilling his early ambition of becoming a writer, Skinner wrote several popular books, including *Walden Two* (first published in 1948), which described a highly regulated utopian society in which socially desirable behaviors would be maintained through the same kind of training regimens Skinner applied to his pigeons and rats. (Transmission of socially desirable behaviors is also discussed in [Chapter 11](#) of the book you are now reading.) In another best-selling book, *Beyond Freedom and Dignity* (first published in 1971), Skinner advocated an extreme form of behaviorism, often called **radical behaviorism**, in which he asserted that consciousness and free will are illusions. Skinner argued that humans, like all other animals, function by blindly producing learned responses to environmental stimuli.



What do the titles of these works seem to say about Skinner's interests and beliefs?

Description

The photo on the left shows the book cover that reads, *Walden Two*, by B. F. Skinner. The photo on the right shows the book cover that reads, *The million-copy bestseller by the author of Walden Two. A stunning new plan to alter human behavior. Beyond Freedom and Dignity*, by B. F. Skinner. A text below reads, open quotes *If you plan to read only one book this year, this is probably the one you should choose!* Close quotes *The New York Times*.

radical behaviorism

An extreme form of behaviorism championed by B. F. Skinner and holding that consciousness and free will are illusions and that even so-called higher cognitive functions (e.g., human language) are merely complex sets of stimulus–response associations.

By the middle of the twentieth century, Skinner was the most famous psychologist in the world. He continued promoting radical behaviorism right up until the night of his death in 1990; but by that time, mainstream psychology had moved past the confines of strict behaviorism to focus on the very kinds of mental events, and related brain activity, that Skinner and his fellow behaviorists had fought so hard to discredit.

Interim Summary

- René Descartes was strongly allied with the nature (or nativist) camp and believed that we inherit our talents and abilities. He described a highly mechanistic approach to behavior that

included a detailed reflex arc from sensory stimulus to motor response.

- Borrowing conceptual frameworks from the physical sciences, John Locke believed children arrive into the world as blank slates (*tabulae rasae*) to be imprinted upon by experience and learning.
- John Watson, a leading early proponent of behaviorism, believed that all behavior is learned and the product of our environment.
- B. F. Skinner combined his talents as a writer and an experimental psychologist to develop modern methods for studying the details of how animals learn associations between responses and consequences.

1.3 Comparing Humans to Other Animals

Both Watson and Skinner did research primarily on rats and pigeons, and from their laboratory work with these animals, they extrapolated principles of learning and memory that they believed applied equally well to people. Even so, they both recognized that there are important differences between laboratory animals and people.

How unique humans are within the animal kingdom is a question that has been posed throughout the history of psychology. Plato and other classical Greek philosophers took one extreme view: they believed that humans are unique among living things because they possess an everlasting soul. Aristotle, in contrast, argued that humans exist in a continuum with other animals and that the ability to reason is their sole distinguishing feature. Renaissance philosophers tended to side with Plato, bolstered by the Church-sponsored view that humankind was created in God's image. For example, Descartes believed that humans and animals are fundamentally different, just as he believed that mind and body are separate.

But by the early 1800s, this view of humans as being fundamentally different from animals was beginning to meet serious challenge. European naturalists had begun to collect and study a wide variety of plants and animals from around the world. The facts these scientists uncovered and the theories they developed upended many long-held beliefs about who we are, where we come from, and how similar we might be to other animals. These new perspectives on the relationship between animals and humans would profoundly affect the study of natural history, as well as all future studies of the psychology of learning and memory.

Erasmus Darwin (1731–1802), the personal doctor to King George III of England, was an early and vocal proponent of the theory of [evolution](#), which states that living species change over time, with new traits or characteristics emerging and being passed from one generation to the next. With sufficient time, Darwin argued, one species could evolve so far that it would come to constitute an entirely different species from its ancestor ([Darwin, 1794](#)). It was many years later, however, that Erasmus's grandson Charles developed a theory of how this evolutionary process might take place—and in doing so made the term *evolution* synonymous with the Darwin family name.

evolution

The change that species undergo over time, with new traits or characteristics being passed from one generation to the next;

natural selection is one mechanism by which evolution occurs.

Charles Darwin and the Theory of Natural Selection

Charles Darwin (1809–1882), Erasmus's grandson, was himself the son of a prosperous doctor, while his mother descended from the wealthy Wedgwood family of ceramic ware fame. His family's financial position meant that he did not have to work for a living. Instead, what Darwin most enjoyed was to walk through the English countryside collecting and cataloging animals.

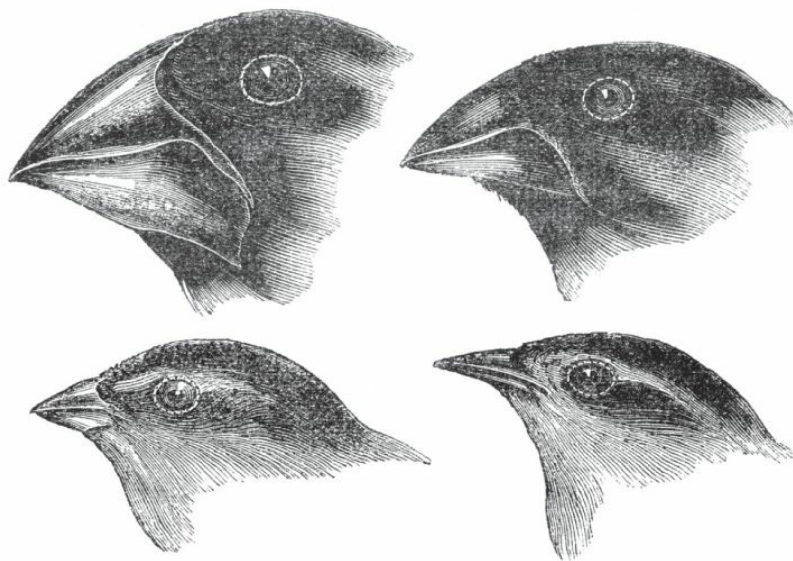


Eileen Tweedy/REX/Shutterstock

Charles Darwin

In 1831, at age 22, with no career direction other than his amateur interest in natural history, Charles Darwin accepted an offer to accompany the captain of the *HMS Beagle* on an expedition to chart the coast of South America. In South America, Darwin encountered an abundance of

previously unknown species, many on the Galápagos Islands, an isolated archipelago off the coast of Ecuador. Of particular interest to Darwin were the many species of birds he observed, especially the finches—of which he identified at least 14 varieties, each on a different island ([Figure 1.4](#)). On one island that had plentiful nuts and seeds, the finches had strong, thick beaks that they used to crack open nuts. On another island, with few nuts but plenty of insects, the finches had long, narrow beaks, perfect for grabbing insects from the crevices of tree bark. Each isolated island in the archipelago was populated by a different kind of finch, with a beak ideally suited to that island's distinct habitat. In his account of the trip, Darwin wrote that “one might really fancy that from an original paucity of birds in this archipelago, one species had been taken and modified for different ends” ([Darwin, 1845](#)). Charles Darwin, like his grandfather Erasmus, was convinced that life on earth was evolving, not immutably fixed.



Universal History Archive/REX/Shutterstock

FIGURE 1.4 Finches of the Galápagos Islands Note the strong, heavy beak of the bird at the upper left (good for cracking nuts) and the long, narrow beak of the bird at the lower right (good for grabbing insects from cracks in bark).

Description

The first finch has a thick and strong beak. The second finch has a flat beak. The third finch has a flat, but a smaller beak. The fourth finch has a narrow beak.

Darwin's most important legacy was his theory of [natural selection](#), which proposed a mechanism for how evolution occurs ([Darwin, 1859](#)). He argued that species evolve when they

possess a trait that meets three conditions ([Table 1.1](#)). First, the trait must be *inheritable*, meaning it can be passed from parent to offspring. (Keep in mind that genes—the carriers of inherited traits—had not yet been discovered in Darwin’s time.) Second, the trait must *vary*, having a range of forms among the individual members of the species. Third, the trait must make the individual more *fit*, meaning that it must increase reproductive success—that is, increase the chance that the individual will survive, mate, and reproduce, passing on the trait to its offspring. This, in turn, will make the offspring more fit, increasing their chances of surviving and passing on the trait. Over time, natural selection (sometimes called “survival of the fittest”) causes the trait to spread through the population. This, Darwin argued, was the underlying mechanism by which species evolve.

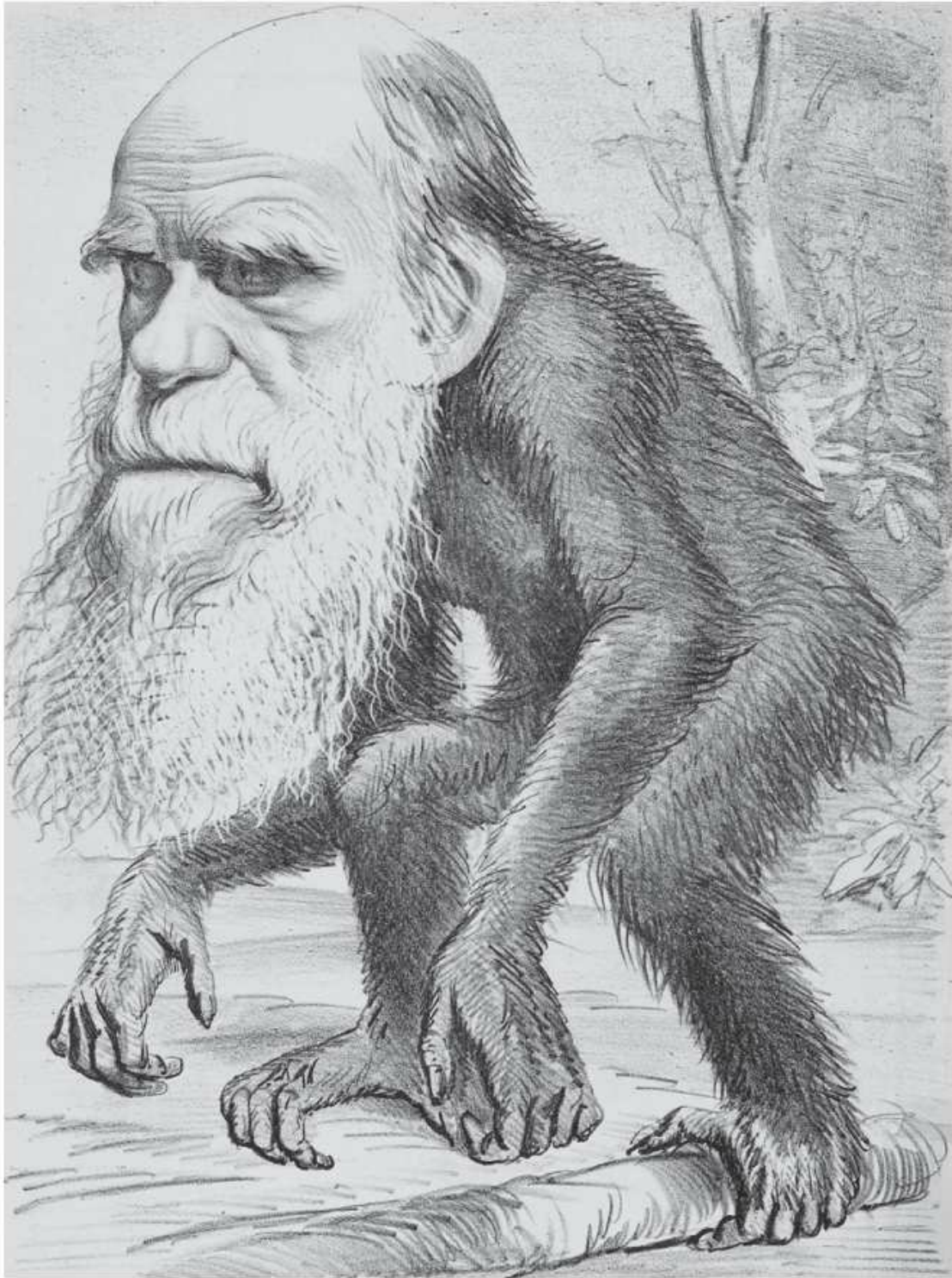
TABLE 1.1 Darwin’s three criteria for traits to evolve through natural selection

Criterion	Finches
1. Inheritable trait	Beak shape
2. Natural variability	Thin or thick
3. Relevance to survival	Improves access to insects (thinner beak) or ability to crack nuts (thicker beak)

natural selection

A proposed mechanism for evolution, also known as “survival of the fittest,” which holds that species evolve when there is some trait that is inheritable, varies naturally across individuals, and increases an individual’s “fitness,” or chance of survival and reproductive success.

Darwin tinkered with his ideas for 20 years. Finally, in 1859, he published *On the Origin of Species by Means of Natural Selection, or the Preservation of Favoured Races in the Struggle for Life*, more commonly known by its abbreviated title, *The Origin of Species*. Darwin’s book became a best seller, was translated into many languages, and ignited a major public controversy that resulted in thousands of reviews, articles, and satires. Why the uproar? Darwin’s view of natural selection upset many people’s belief that there is an important distinction between “man and beast.” Theologians were alarmed because the idea that humans and apes evolved from a common ancestor seemed to challenge the biblical doctrine that people were created by the hand of God, in God’s own image. *The Origin of Species* is among the most controversial scientific books ever written.



Hulton Archive/GraphicaArtis/Getty Images

Darwin was the subject of many personal attacks. What inspired this caricature, published in *Hornet* magazine on March 22, 1871?

What are the implications of Darwin's work for the psychology of learning and memory? Darwin argued that behavioral traits could evolve through the same process of natural selection as physical traits ([Darwin, 1872](#)). Learning has enormous value for survival, allowing organisms to adapt to a changing and variable world. Thus, organisms with more capacity for learning and memory are more fit—better able to survive and more likely to breed and pass along their inherited capacities to offspring. Notice that the content of what is learned is not passed on: learned knowledge is an acquired trait, which cannot be inherited. What can be inherited is the *capacity* or *ability* for learning and memory. Further discussion of how genetics influences learning and memory appears in [Chapter 12](#).

The Neo-Behaviorism of Edward Tolman

While Darwin was arguing that humans and animals share the ability to evolve, researchers such as Edward Tolman (1886–1959) were arguing that humans and other animals are not limited to simple stimulus–response reflexes but, in addition, are able to operate purposefully to achieve desired goals. As a college student at the Massachusetts Institute of Technology (MIT), Tolman originally planned to pursue a career in chemistry. However, during his senior year, he read William James's *Principles of Psychology* and was so inspired that he decided instead to pursue a graduate degree in psychology.



The Drs. Nicholas and Dorothy Cummings Center
for the History of Psychology, The University of
Akron

Edward Tolman

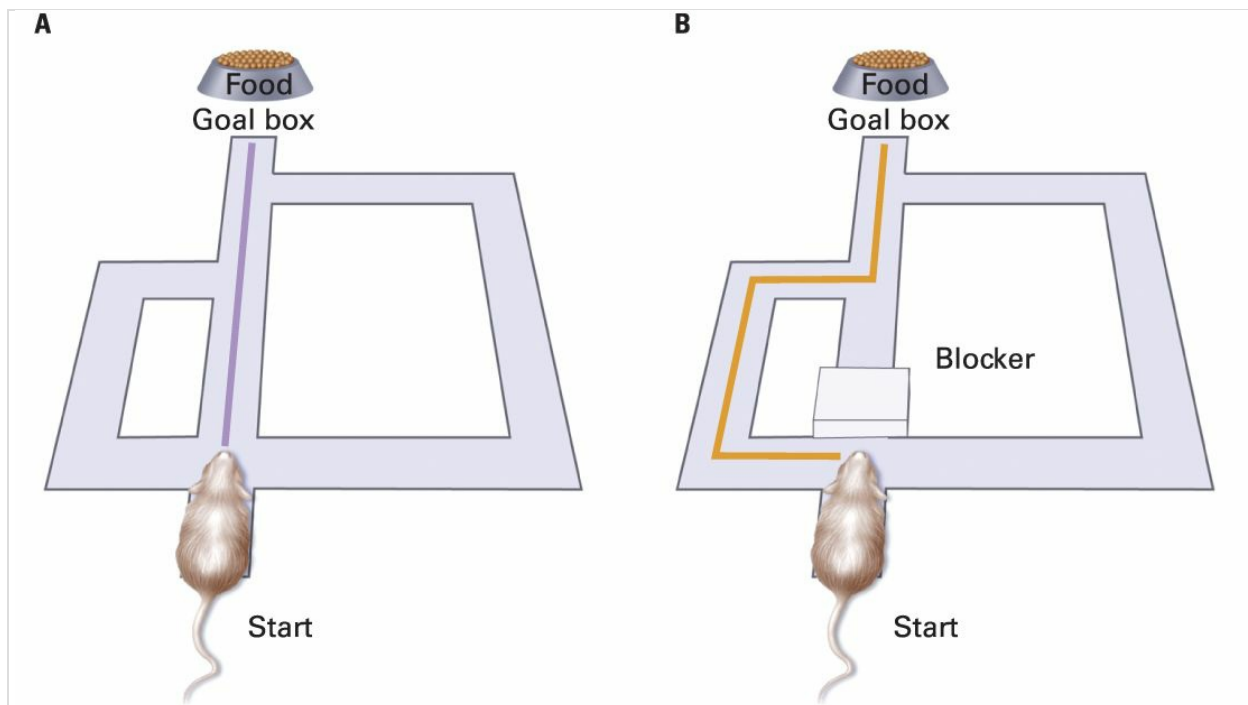
Tolman began building a series of rat mazes for the study of learning, much as Thorndike and

Watson had done before him. But in contrast to Watson, Pavlov, and Skinner, who had argued for a purely mechanical approach that described rat learning as the formation of associations between stimuli and responses, Tolman was convinced his rats were learning something more. He believed that rats have goals and intentions, much as humans do, such as the goal of finding the exit and seeking food. Rats, he argued, are like humans in that they are intrinsically motivated to learn the general layout of mazes by forming what he called a **cognitive map**, an internal psychological representation of the spatial layout of the external world ([Tolman, 1948](#)). “Behavior reeks of purpose” was Tolman’s well-known and oft-repeated maxim ([Tolman, 1932](#)).

cognitive map

An internal psychological representation of the spatial layout of the external world.

In one series of studies, Tolman showed the value of cognitive maps for understanding how rats can apply what they have learned in novel situations. Rats, he showed, are able to find food in mazes by using alternative routes if their preferred route is blocked, as shown in [Figure 1.5](#) ([Tolman, 1948](#)). They can also find their way to the goal if they are started from a novel position in the maze rather than the usual starting point. None of this could be explained by the learning of simple stimulus–response associations favored by Pavlov and Skinner, and it suggested that rat learning was far more like human learning and exploration than the simple models proposed by these leading animal learning theorists.



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FIGURE 1.5 Cognitive maps in rats Tolman believed that rats form cognitive maps, internal representations of the layout of the world. (A) In one experiment, rats placed at the start of a maze learned to run directly to a goal box where food was provided; the purple line shows the rats' route. (B) If the preferred route was blocked, rats could easily find an effective alternative route (orange line); this indicates that they had information about the spatial layout of the maze.

Description

The illustration on the left is labeled A. It shows a rectangular box-shaped maze. A rat, placed at the starting of the maze, is labeled Start and a bowl of food, placed at the other end of the maze, is labeled Goal box. A straight line is drawn meeting both the ends.

The illustration on the right is labeled B. It shows a rectangular box-shaped maze, with a blocker in front of a rat. The rat, placed at the starting of the maze, is labeled Start. A bowl of food, placed at the other end, is labeled Goal box. An alternative line is drawn meeting both the ends. The lines indicate the path the rat travels.

Tolman even showed that rats can form cognitive maps in the absence of any explicit reward (such as food). He allowed some rats to freely explore a maze (like the one in [Figure 1.5](#)) with no food in it, for several days. Later, when he placed these rats in the maze with a food reward at one location ("goal box"), they learned to find the food much faster than rats not previously exposed to the maze and almost as fast as rats that had been explicitly trained to find the food in the goal box. These studies are described in more detail in [Chapter 3](#) (see especially [Figure 3.7](#)). This, Tolman argued, showed that during their free exploration, the rats were learning a cognitive

map that they could exploit later. He called the phenomenon **latent learning**, meaning learning that takes place even when there is no specific motivation to obtain or avoid a specific consequence, such as food or shock ([Tolman, 1932](#)). Tolman reasoned that such latent learning is a natural part of the everyday life of both rats and humans. The idea of latent learning challenged a strict behaviorist assumption that all learning reflects stimulus–response associations. The effects of latent learning are discussed in [Chapters 3](#) and [6](#).

latent learning

Learning that is unconnected to a positive or negative consequence and that remains undetected (latent) until explicitly demonstrated at a later stage.

Emphasizing the importance of internal representations of the environment and utilizing concepts such as purpose and intent that are merely inferred, not directly observable, Tolman broke away from the stricter confines of behaviorist dogma, all the while satisfying the behaviorists' high standards of experimental control and methodological rigor. For this reason, Tolman is often referred to as a *neo-behaviorist*. His influential theoretical and experimental research—though at odds with many of his behaviorist contemporaries—laid the foundation for studies of animal and human learning that blended cognitive and behavioral perspectives.

[Interim Summary](#)

- Most early philosophers assumed that humans are quite distinct from and innately superior to animals, but the proponents of evolution, such as Erasmus Darwin and Charles Darwin, showed how similar we are.
- Charles Darwin argued that species evolve when they possess a trait that meets three conditions: it is inheritable, it varies, and it increases the fitness of the individual.
- Edward Tolman believed that rats have goals and intentions, much as humans do, such as the goal of finding the exit to a maze and obtaining food. Rats, he argued, are like humans in that they are intrinsically motivated to learn the general layout of mazes by forming what he called a cognitive map, an internal psychological representation of the spatial layout of the external world.

1.4 Formal Models and Universal Laws

Throughout the history of studies on learning and memory, philosophers and psychologists have borrowed methods and metaphors from physics, chemistry, and other scientific fields to enhance their understanding. We will trace here almost a century of such fortuitous cross-disciplinary borrowing. In current research, most psychologists hold themselves to the same rigorous principles of experimental methodology adhered to by scientists in other disciplines; if psychologists want their work to be taken seriously, they have to pay close attention to experimental design and analysis.

Hermann Ebbinghaus and Human Memory Experiments

Hermann Ebbinghaus (1850–1909), a contemporary of William James, conducted the first rigorous experimental studies of human memory. After earning his PhD, Ebbinghaus lived an itinerant life in Europe and England, traveling, attending occasional seminars, and working for short periods as a teacher and private tutor. One day, browsing at a bookstall, he came across a book by a German physicist, Gustav Fechner (1801–1887), that described the science of human perception. Fechner showed that there are highly predictable regularities in how people perceive variations in physical stimuli, such as changes in the brightness of a light or the weight of a ball. The book illustrated how a simple mathematical equation could describe the relationship between the physical world and the psychological world. Captivated by these ideas, Ebbinghaus proposed that the psychology of memory could also become a rigorous natural science, defined by precise mathematical laws.



Bettmann/Getty Images

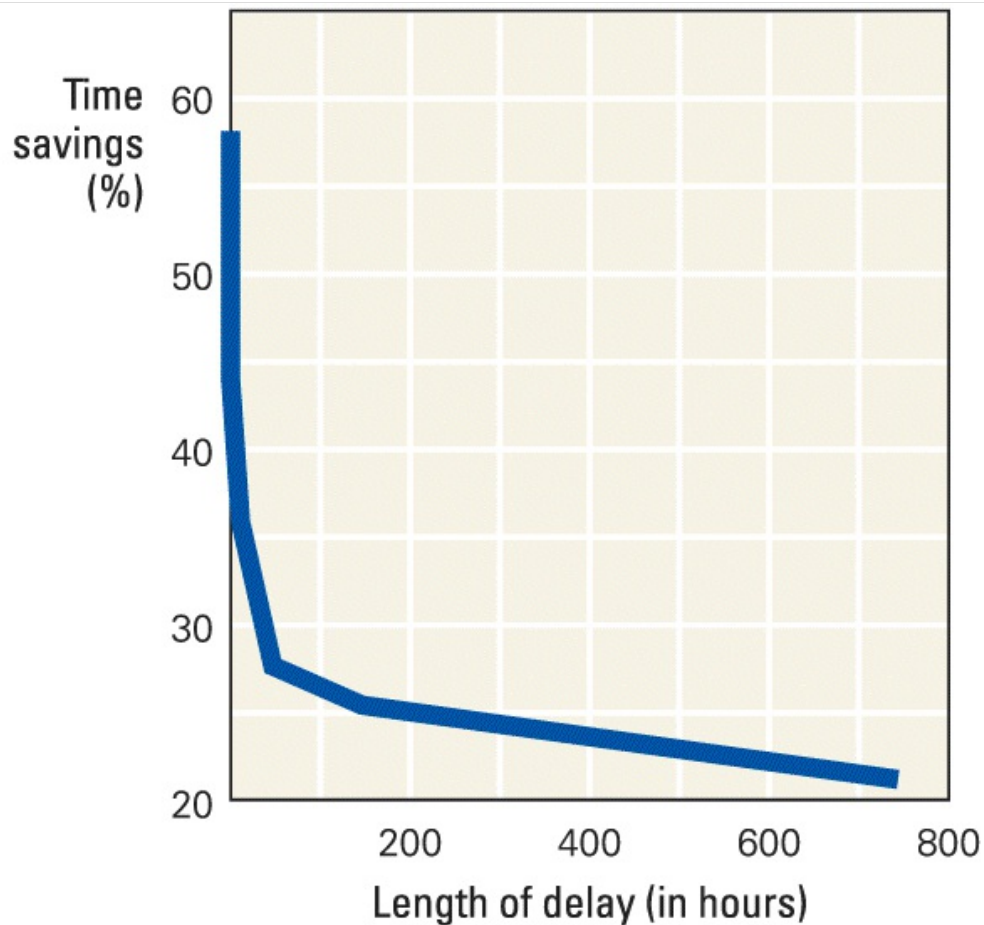
Hermann Ebbinghaus

Unlike many of the other scientists discussed in this chapter, Ebbinghaus was not a wealthy man. Unable to afford to pay anyone to participate in his research, he did his studies using himself as the only test participant. Despite this limitation, his work laid the foundation for all future experimental studies of human memory; in fact, Ebbinghaus is often considered to be the father

of modern memory research.

Ebbinghaus sought mathematical equations to explain how memories are acquired and how they fade. Early on, he realized that if he studied lists of real words, his data would be strongly affected by the fact that he was more familiar with some words than others. To avoid this problem, he used three-letter nonsense words, such as BAP, KEP, and DAK, which would be unfamiliar to him. In one of his experiments, Ebbinghaus read a list of 20 words out loud to himself, put the list away for a while, and then tried to remember as many words as possible. Afterward, he checked which words he missed, reviewed the list, and tried again. He repeated this process until he could remember all 20 words from the original list. This experiment incorporates the four key stages—*learning*, *delay*, *test*, *relearning*—that would represent the basic methodology for human memory experiments for years to follow. Further examples of these kinds of studies are discussed in [Chapter 7](#).

Ebbinghaus was especially interested in [forgetting](#)—that is, in how memory deteriorates over time. He measured forgetting by examining how long it took him to relearn a previously learned list. If it initially took him 10 minutes to learn the list and later took only 6 minutes to relearn the same list, Ebbinghaus recorded a “time savings” of 4 minutes, or 40% of the original learning time. By testing himself at various intervals after learning (with different lists each time), Ebbinghaus was able to plot a [retention curve](#), which measures how much information is retained at each point in time following learning. [Figure 1.6](#) shows an example of this, graphing the percentage savings in time for relearning after delays of different lengths between the initial learning and relearning ([Ebbinghaus, 1885/1964](#)).



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FIGURE 1.6 Ebbinghaus's retention curve These experimental data show the percentage savings in time for relearning a list of words as a function of the delay between learning and relearning. Ebbinghaus's early study demonstrated that retention drops quickly in the first few days (up to about 100 hours for the task shown here) and then tapers off more slowly with increasing delays.

Data from [Ebbinghaus, 1885/1964](#).

Description

The horizontal axis plots Length of delay in hours and the vertical axis plots Time Savings in percent. The data in the graph are as follows. (50, 57). (50, 50). (50, 40). (51, 35). (75, 27). (100, 26). (130, 25). (200, 25). (300, 24). (400, 23). (500, 22). (600, 21.5). (700, 21). (750, 20.5). All data are approximate.

forgetting

The loss or deterioration of memory over time.

retention curve

A graph showing forgetting or relearning as a function of time since initial learning.

As you can see in [Figure 1.6](#), there is a strong time savings (nearly 100%) if the delay between learning and relearning is short. But as the delay grows longer, to about 100 hours (approximately 4 days), the savings declines to about 27%. The retention curve also illustrates that most forgetting occurs early on: if a memory can survive the first few hours after learning, there is little additional forgetting. Thus, Ebbinghaus showed a savings of about 25% after 200 hours, and this dropped only to 20% after 750 hours. In other studies, Ebbinghaus showed that shorter lists were easier to remember than longer lists. He also demonstrated that increasing the amount of initial practice improved later recall.

Forgetting is a normal part of life for all of us. But what if you forgot everything new that happened to you? What if you lived entirely in the present, forgetting every experience almost immediately after it occurred? People like this do exist, and one of them is named Clive Wearing. You can learn more about his memory struggles in “[Learning and Memory in Everyday Life](#)”. After reading this feature, you might ask yourself what [Figure 1.6](#) would look like if the participant were Clive Wearing rather than Hermann Ebbinghaus.

LEARNING AND MEMORY IN EVERYDAY LIFE

What If You Could Form No New Memories?

At age 46, Clive Wearing had it all. He was a well-known, highly regarded symphony conductor; he was handsome, charming, and witty; and he was deeply in love with his wife, Deborah. Then his memory was stripped from him. Clive had developed a rare condition in which a virus that usually causes nothing more serious than cold sores invaded his brain. His brain tissue swelled, crushing against the confines of his skull. Although most patients die when this happens, Clive survived, but his brain remained significantly damaged.



Jiri Rezac/Polaris

Clive Wearing with his wife, Deborah.

When Clive awoke in the hospital, he had lost most of his past. He could recognize Deborah but could not remember their wedding. He knew he had children but could not remember their names or what they looked like. He could speak and understand words, but there were huge gaps in his knowledge. On one test, when shown a picture of a scarecrow, he replied: “A worshipping point for certain cultures.” Asked to list famous musicians, he could produce four names: Mozart, Beethoven, Bach, and Haydn. Conspicuously absent from this list was Orlande de Lassus; Clive had been the world expert on this sixteenth-century composer ([Wilson & Wearing, 1995](#)).

But Clive Wearing had not lost just the past: he had also lost the present. For a few seconds he would retain consciousness of whatever he happened to be experiencing, and then the information would melt away without forming even a temporary memory. During his stay in the hospital, he had no idea where he was or why he was surrounded by strangers. Whenever he caught sight of Deborah after a short interval—say, if she had left the room for a few minutes—he would run to her and kiss her joyously, as if she had been absent for years. A few minutes later, he would catch sight of her again and stage another passionate reunion.

In short, Clive now lived only “in the moment,” caught in an endless loop of reawakening. His numerous journal entries show his desperate efforts to make sense of what he was experiencing: “7:09 a.m.: Awake. 7:34 a.m.: Actually finally awake. 7:44 a.m.: Really perfectly awake ... 10:08 a.m.: Now I am superlatively awake. First time aware for years. 10:13 a.m.: Now I am overwhelmingly awake.... 10:28 a.m.: Actually I am now first time awake for years....” Each time he added a new entry, he might go back and scratch out the previous line, angry that a stranger had written misleading entries in his journal.

Yet even when Clive knew nothing else, he knew that he loved his wife. Emotional memory—love—survived when almost everything else was gone. And he could still play the piano and conduct an orchestra so competently that a nonmusician wouldn’t suspect anything was wrong with Clive’s mind. Those specialized skill memories survived, along with more mundane skills, such as making coffee or playing card games. And although Clive was unable to consciously learn new facts, he could acquire new habits through repeated practice. After moving to a nursing home, he eventually learned the route from the dining hall to his room, and when prompted to put on his coat for his daily walk past the local pond, he would ask if it was time to go feed the ducks ([Wilson & Wearing, 1995](#)). Clive’s memory was more like an imperfectly erased blackboard than a blank slate.

Clive Wearing’s tragic case makes two important points. The first is the unrivaled importance of learning and memory to our lives. Most of the time, we take for granted our memories of who we are and what we know. When these are stripped away, life becomes a series of unrelated moments, isolated from past and future, like the momentary disorientation we all experience upon awakening.

The second point is that there are many different kinds of memory; it is not a single, cohesive process. And as Clive’s case demonstrates, some kinds of memory can be damaged while others are spared. Normally, the different kinds of memory function together seamlessly, and we have no awareness of whether a given instance of learning has been preserved as a fact, a habit, a skill, or an emotion. But this cohesion is in many ways an illusion. By confronting the limits of this illusion, we can begin to understand how memory works, both in healthy people and in individuals whose memory has broken down. [Chapters 7](#) through [9](#), the “Memory Module” of this book, cover fact and autobiographical memory, skill memory, working memory, and emotional memory; [Chapter 7](#) goes into further specifics about amnesic patients like Clive Wearing.

In designing his experiments, Ebbinghaus started with a prediction, or [hypothesis](#), a supposition made on the basis of limited evidence as a starting point for further investigation. To test this

hypothesis, he began carefully manipulating an independent variable (say, the length of the delay between learning and relearning) and measuring the effect on a dependent variable (say, memory retention). This design enabled Ebbinghaus to show a relationship between the independent variable (e.g., length of delay) and the dependent variable (e.g., memory retention).

hypothesis

A supposition made on the basis of limited evidence as a starting point for further investigation.

The major limitation of Ebbinghaus's studies was that they were conducted with just one participant, Ebbinghaus himself. There are several reasons why such self-experimentation is problematic and would not meet modern scientific standards for research. For one thing, what if Ebbinghaus's memory was different from that of most other people? In that case, the results of his experiments would tell us a lot about Ebbinghaus but would not be applicable to other people. This is why modern research on memory usually involves testing a large number of people.

A second problem is that, because he was the experimenter as well as the participant, Ebbinghaus knew which variables were being manipulated. If he believed, for example, that longer lists were harder to learn, then this belief might subtly influence his performance as the experimental subject, causing him to take longer to learn those lists. This problem is sometimes called **subject bias**. To avoid such problems, modern studies of memory employ a **blind design**, which means the participant does not know the hypothesis being tested or the variables being manipulated. There is also a corresponding problem of **experimenter bias**, which means that even a well-meaning experimenter might influence the outcome (for example, by implicitly encouraging the participant to respond in an expected manner).

subject bias

The influence a subject's prior knowledge or expectations can have (consciously or unconsciously) on the outcome of an experiment.

blind design

An experimental design in which the participants do not know the hypothesis being tested or whether they are, for example, receiving an experimental treatment or a placebo.

experimenter bias

The influence an experimenter's prior knowledge or expectations can have (consciously or unconsciously) on the outcome of an experiment.

Experimenter bias can be avoided by employing a **double-blind design**, in which neither the

participant nor the experimenter knows which participant is getting which treatment or intervention. Testing of experimental medications is a common modern example of the double-blind strategy. Patients receive either the test drug or a **placebo** (an inactive substitute that looks just like the real drug), and neither the patients nor the doctors know who is receiving which substance; only the people analyzing the results (who never interact directly with the research participants) know which is which.

double-blind design

An experimental design in which neither the experimenters nor the subjects know group assignment.

placebo

An inactive substance, such as a sugar pill, that is administered to one group of participants in an experiment to compare against the effects of an active substance, such as a drug.

Despite the imperfections in his experimental designs, Ebbinghaus led the way in the use of scientific experimentation to study learning and memory. The vast majority of studies of human memory conducted today owe their methodology to the early and influential work of Hermann Ebbinghaus.

TEST YOUR KNOWLEDGE

Who's Who in the History of Learning and Memory?

Below is a (slightly tongue-in-cheek) review of some of the major researchers and ideas covered in this chapter. See if you can fill in the blanks with the missing names. (*Answers appear at the end of the chapter.*)

1. Old _____ was a Greek
Who thought about association.
_____, the dualist, liked to speak
Of mind-and-body separation.
2. To _____, a baby's mind was blank,
As all empiricists have said.
Nativists called him a crank,
Believing knowledge is inbred.
3. _____'s models of the mind
Had features linked together,
Updating Greeks from ancient times
And going them one better.
4. _____ learned nonsense words;
Dogs learned to drool for _____.
_____ studied food rewards
(And coined "effect, the law of").

Clark Hull and Mathematical Models of Learning

Clark Hull (1884–1952) devoted his career to developing mathematical equations by which he hoped to describe the relationships among the factors that influence learning. Hull was born on a farm near Akron, Ohio, and his early life was marked by life-threatening illness. He survived an attack of typhoid fever but sustained lasting brain damage, which caused memory difficulties that plagued him for the rest of his life. He also survived a bout of polio that left him paralyzed in one leg and dependent on crutches to walk.



Office of Public Affairs, Yale University,
Photographs of Individuals (RU686).
Manuscripts and Archives, Yale University
Library.

Clark Hull

In Hull's day, the new doctrine of behaviorism claimed that all behavior could be understood as a set of connections between stimuli and responses. When Pavlov's dogs heard the doorbell, they salivated (doorbell → salivation); when Watson's rats entered the maze, they made a series of motor-habit responses (maze entry → turn left, turn right, and so on). Such learning is often called *stimulus-response learning*, abbreviated as *S → R learning*. Of course, the behaviorists

acknowledged that the real world is complicated and that other factors might affect the response. For example, Pavlov's dogs might salivate to the doorbell only if they were hungry. Still, the behaviorists believed that if you could specify all the existing factors, you ought to be able to predict exactly whether and when a stimulus would provoke an animal to make a response.

Hull set himself the goal of developing a comprehensive mathematical model of animal learning that would predict exactly what an animal will learn in any given situation. Much as Einstein had recently shown that a single equation, $E = mc^2$, could explain the complex relationship between energy (E), mass (m), and the speed of light (c), Hull hoped to find a similarly powerful equation to relate all the key factors contributing to a learning experience. The variables that Hull entered into his equations included the number of learning trials, the frequency of reward, the spacing between trials, the intensity of the stimulus cues, the animal's motivation for reward, and the incentive value (desirability) of the reward ([Hull, 1943](#)). Hull conducted an intensive program of research on learning in animals and humans, seeking to test and refine his mathematical models. One measure of a model's value is its ability to serve as a basis for experimental research; in this regard, Hull's model was a great success. By the 1940s, Hull's work was being cited in 70% of all scientific papers on learning published in the major journals of the time ([Spence, 1952](#)).

Although Hull's equations were influential in their era, their specifics are no longer considered relevant today. Hull's models have been abandoned because modern psychologists have despaired of ever being able to reduce all the factors governing learning into a single equation, as Hull had hoped to do. Nevertheless, Hull's many students and followers (often called neo-Hullians) carried on toward a more modest goal: to develop mathematical equations to describe basic kinds or components of learning. ([Chapter 4](#) presents one of the most enduring neo-Hullian learning theories: the Rescorla–Wagner rule, which describes some factors governing classical “Pavlovian” conditioning.) Neo-Hullian researchers showed that learning indeed follows reliable, predictable patterns and pointed the way toward an understanding of how the same basic patterns govern learning in humans as in other animals.

W. K. Estes and Mathematical Psychology

William K. Estes (1919–2011) and his colleagues established a new subdiscipline of psychology, [mathematical psychology](#), which used mathematical equations to describe the laws of learning and memory and demonstrated how quantitative approaches can be applied to observable

behaviors in order to understand, and formally model, mental functions. Estes began his graduate studies under the tutelage of Skinner during the early 1940s. Together, they developed a new method for studying classical “Pavlovian” conditioning of fear in rats. Within a few years, their procedure became one of the most widely used techniques for studying animal conditioning, and it is still in use today (and further described in [Chapter 4](#)). Learning about fear and other emotions has since become another important subfield of learning and memory research (as discussed in [Chapter 10](#)).



George Estes

W. K. Estes

mathematical psychology

A subfield of psychology that uses mathematical equations to describe the laws of learning and memory.

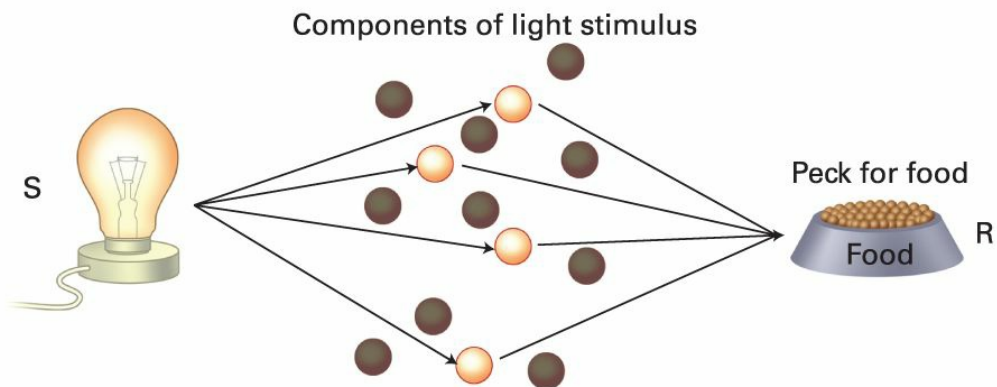
Estes completed his PhD during World War II and was called into military service. Stationed in the Philippines as the commandant of a prisoner-of-war camp, he had time to read mathematics books his wife sent from home. When the war ended, Estes returned to the United States and to the study of psychology. Much to Skinner's dismay, Estes soon began to stray from his mentor's strict behaviorism. He began to use mathematics to describe mental events that from behavioral data could only be inferred indirectly, an approach quite unacceptable to behaviorists. Years later, in his autobiography, Skinner bemoaned the loss of Estes as a once-promising behaviorist, speculating that Estes's preoccupation with mathematical models of unobservable mental events was a war-related injury, resulting perhaps from too much time in the hot Pacific sun ([Skinner, 1979](#)).

Estes built on Hull's mathematical modeling approach to develop new methods for interpreting a wide variety of learning behaviors ([Estes, 1950](#)). Most learning theorists of that era, including Hull, assumed that learning should be viewed as the development of associations between a stimulus and a response. For example, suppose that a pigeon is trained to peck whenever it sees a yellow light in order to obtain a bit of food. Hull assumed that this training caused the formation of a direct link between the stimulus and the response, so that later presentations of the yellow light evoked the peck-for-food response ([Figure 1.7A](#)).

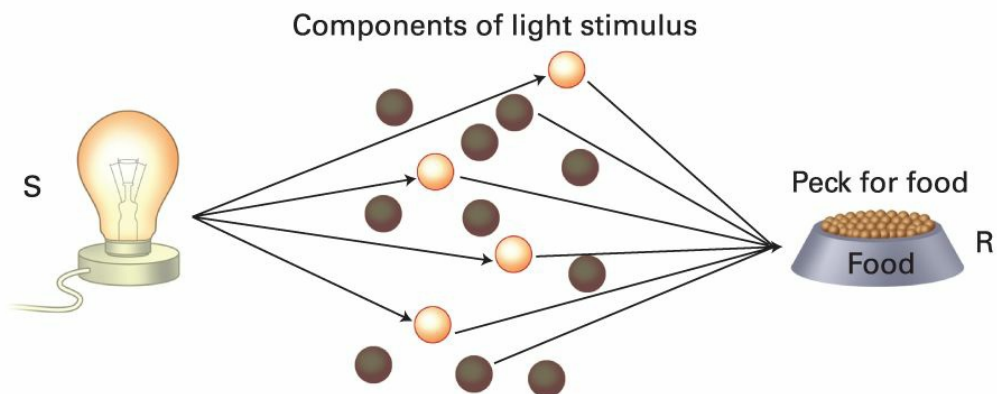
A Hull: Direct S–R associations



B Estes: Stimulus sampling theory, first trial



C Estes: Stimulus sampling theory, second trial



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

FIGURE 1.7 Stimulus–response models How does a stimulus (S) become associated with a response (R)? (A) Hull assumed that a direct link was formed between a stimulus (such as a yellow light) and a learned response (such as, in pigeons, pecking for food). (B) Estes proposed an intervening stage, in which a random sample of elements representing various components of the stimulus “yellow light” are noticed, activating a pattern of connections in the brain; those elements and pattern are then associated with the response. (C) On a different trial, a different random subset of elements making up the stimulus are noticed and associated with the response. Over time, with many such random samples, most elements that could potentially be recognized as aspects of the stimulus become associated with the response. At this point, when a random sample of elements are noticed upon presentation of the light, most of them are already linked with the response.

Description

Each illustration has a bulb marked S and a bowl labeled, Peck for food and marked R.

The first illustration is labeled, A. Hull: Direct S–R associations. A one-directional arrow starts from S and points toward R.

The second illustration is labeled, B. Estes: Stimulus sampling theory, first trial. Various components of the stimulus, S is associated with the response R. The reaction is labeled Components of light stimulus. Random subsets of S-R pairs are formed.

The third illustration is labeled, C. Estes: Stimulus sampling theory, second trial. Various components of the stimulus, S is associated with the response R. The reaction is labeled Components of light stimulus. Random subsets of S-R pairs are formed.

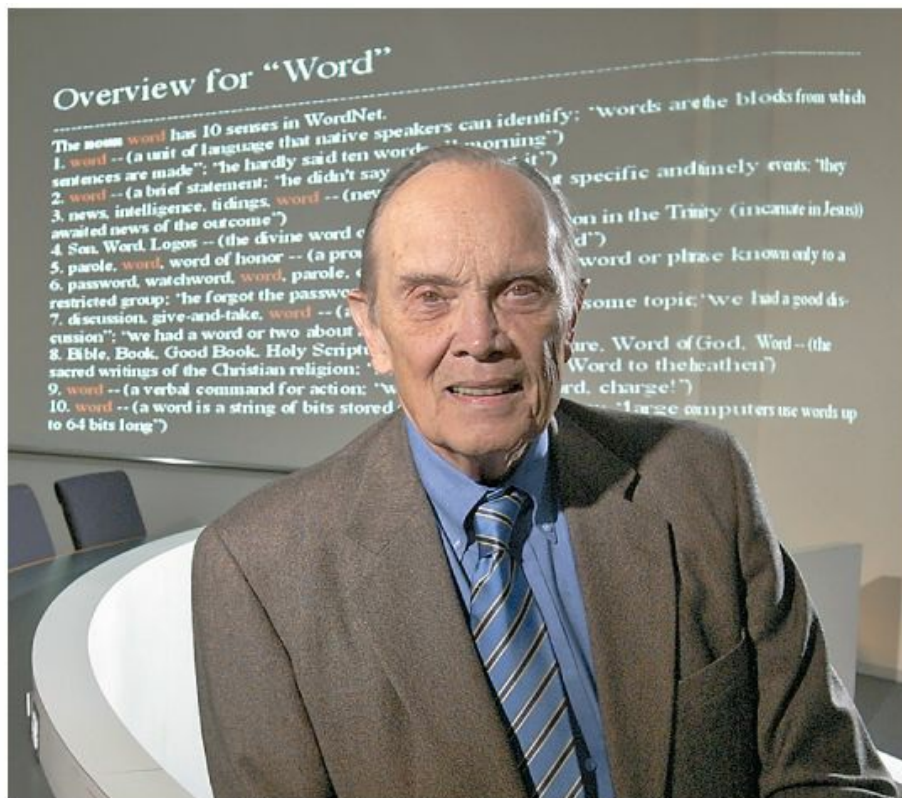
Estes, however, suggested that what seems to be a single stimulus, such as a yellow light, is really a collection of many different possible elements making up the yellow light, with each element being some small simple feature or component of a feature associated with yellow light. According to Estes, only a random subset of these elements are noticed (or “sampled,” in Estes’s terminology) on any given training trial ([Figure 1.7B](#)); and only those elements sampled on that particular trial are associated with the food, creating an initial network of connections in the brain. On a different trial, a different subset is sampled ([Figure 1.7C](#)), and those elements are now a network of associations connected with the food. Over time, after many such random samples, most of the elements of the stimulus become associated with the correct response. At this point, any time the light is presented, a random set of its elements will be sampled, but most of them will already be linked with the response.

Estes called his idea *stimulus sampling theory*. A key principle is that random variation (“sampling”) is essential for learning, much as it is essential for the adaptation of species in Charles Darwin’s theory of evolution through natural selection ([Estes, 1950](#)). Estes’s approach gave a much better account than other theories (such as Hull’s) of the variability seen in both animal and human learning, and it helped to explain why even highly trained individuals do not always make the same response perfectly every time: on any given trial, it is always possible that (through sheer randomness) a subset of elements will be sampled (or noticed) that are not yet linked to the response. [Chapter 6](#) shows that Estes’s stimulus sampling theory also explains how animals generalize their learning from one stimulus (e.g., a yellow light) to other, physically similar, stimuli (e.g., an orange light), as Pavlov had demonstrated in the 1920s.

Estes's work marked the resurgence of mathematical methods in psychology, reviving the spirit of Hull's earlier efforts. From his early work in animal conditioning, through his founding role in mathematical psychology, to his later contributions to the study of higher cognitive processes, Estes continued to be a vigorous proponent of mathematical models to inform our understanding of learning and memory. He died in 2011, following a long struggle with Parkinson's disease, a neurological disorder that destroys the brain cells required to learn new habits and that severely impairs movement (see [Chapters 5](#) and [8](#)).

George Miller and Information Theory

Estes and Tolman were not the only investigators to become disillusioned with the strict confines of the behaviorist approach. Other psychologists also began to seek explanations for observations that could not easily be explained by incrementally learned associations between stimulus and response. One of them was George Miller (1920–2012), whose efforts led toward the adapting of information theory to the study of learning and memory.



Jon Roemer

George Miller

During World War II, many Harvard professors worked on problems for the military. As a graduate student at Harvard, Miller was tasked with designing a jamming signal to disrupt German radio communications. This wartime research on spoken communications led Miller to study other questions of speech perception, such as how context affects communication. For example, if a man floundering in the sea shouts, “Help, I’m drowning!” you might understand the message easily, even if the speech is garbled or indistinct; given the context, it is obvious what the man is trying to communicate. On the other hand, if you meet a man on the street and have no prior expectation of what he might be trying to communicate, his speech would need to be much clearer for you to understand his message: is he greeting you, asking for directions, telling you your shoelaces are untied, or soliciting money?

While puzzling over this, Miller read a paper by mathematician and electrical engineer Claude Shannon that described *information theory*, a mathematical theory of communication that provides a precise measure of how much information is contained in a message, based not only on the message itself but also on the listener’s prior knowledge ([Shannon, 1948](#)). For instance, if a friend tells you that Chris, a student in his psychology class, is male, how much information is in the message? It depends on what you already know. If you already know that all the students in his class are male, then the message contains no new information. If, however, you know that the class includes 10 males and 10 females, information theory would say that your friend’s message contains 1 *bit* of information, where a bit is a “binary digit” (1 or 0) that can represent two alternative states (such as 1 = female, 0 = male). If you ask your friend about Chris’s gender, all he has to do is reply “1” (female) or “0” (male); a message composed of a single bit of information is all the answer you need.



Walter Sanders/The LIFE Picture Collection/
Getty Images

Would it surprise you to learn that one of the male students got the highest grade in the class?

Miller's goal was to adapt information theory to psychology. Specifically, could information theory help us understand how people make judgments about the magnitude of any stimulus? How bright is it? How loud? How high in pitch? Miller discovered that people's capacity to make judgments concerning magnitude across a range was limited to about seven alternative values. This is why many rating scales ask you to rate your opinions on a scale of 1 to 7 (as when a survey asks for your opinion of a new product, ranging from 1 = not satisfied, to 7 = totally satisfied).

At the same time, Miller was conducting a seemingly unrelated project to measure the capacity of people's short-term memory for digits: he would read aloud strings of numbers and ask people

to repeat the numbers from memory. Most people, Miller found, could accurately repeat strings of up to 5 to 9 numbers, but almost no one could remember strings of 10 or more digits. The average memory capacity for numbers (sometimes called a *digit span*) seemed to be about 7 digits, plus or minus 2.

Noting that a capacity of seven appeared in both of these contexts—magnitude rating and digit span—Miller used this connection as the humorous title of a paper that summarized both projects: “The Magical Number Seven, Plus or Minus Two” ([Miller, 1956](#)). The paper became one of the most influential and oft-cited papers in cognitive psychology, spurring later research that showed similar limits on the capacity of human working memory for other kinds of information: the “magic number seven” applied not just to digits but to words, pictures, and even complex ideas. Miller’s central message was that the human mind is limited in capacity, that information theory provides a way to measure this capacity, and that similar limits apply to a diverse range of human capabilities. Our working memory capacity and capabilities are the focus of [Chapter 9](#).

TEST YOUR KNOWLEDGE

Borrowing from the Physical and Natural Sciences to Explain the Mind

Throughout the history of learning and memory, many (if not most) of the scientists who made important contributions did so by liberally borrowing from (and sometimes aping) the methods and concepts of the physical and natural sciences. The table below lists seven of these scientists (plus or minus zero) and the source from which they borrowed. Fill out the third column to describe briefly how they applied the borrowed concepts to the study of learning and memory. ([Answers appear at the end of the chapter.](#))

Who ...	Borrowed from ...	To explain or do what?
1. René Descartes	Hydraulic engineering	
2. John Locke	Physics (Newton), chemistry (Boyle)	
3. Hermann Ebbinghaus	Laws of perception	

	(Fechner)	
4. Ivan Pavlov	Telephone exchanges	
5. Edward Thorndike	Evolution by natural selection (Darwin)	
6. Clark Hull	Theory of relativity (Einstein)	
7. George Miller	Information theory (Shannon)	

The Connectionist Models of David Rumelhart

Borrowing a key term from the work of Thorndike (*connection*) and a conceptual framework from William James (see [Figure 1.1](#)), David Rumelhart (1942–2011), a former graduate student of W. K. Estes, developed models of learning and thinking that he described as “connectionist network models” ([Rumelhart & McClelland, 1986](#)). These were mathematical models of networks of connections between simple processing units (nodes) that could be applied to a wide range of problems in understanding human learning and memory.

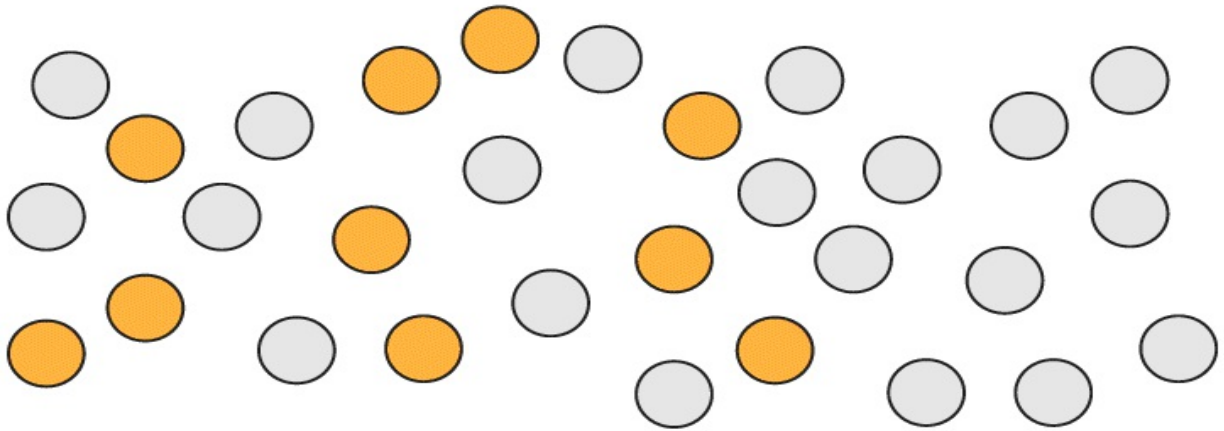


Linda A. Cicero/Stanford News Service

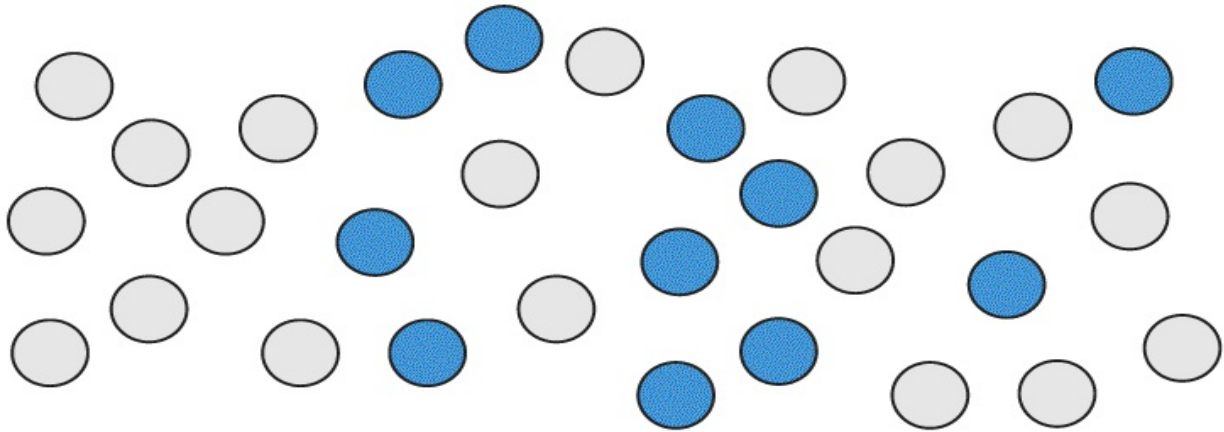
David Rumelhart

In [connectionist models](#), ideas and concepts in the external world are not represented as distinct and discrete symbols but rather as patterns of activity over populations of many nodes. In a connectionist model, a golden retriever might be represented by a pattern of activation across a set of nodes (the orange circles in [Figure 1.8A](#)). A cocker spaniel might be represented by a different pattern of nodes (blue circles in [Figure 1.8B](#)). Such a representation is known as a [distributed representation](#) because the information consists of the activation of many different nodes—that is, it is “distributed” across many nodes rather than represented by only one—in a process similar to what Estes had proposed in his stimulus sampling theory. In a connectionist model, the similarity of spaniels to retrievers emerges because the two breeds activate common elements—the “dog” elements coded as orange-and-blue-striped circles in [Figure 1.8C](#). The importance of distributed representations and shared elements is discussed in more detail in [Chapter 6](#).

A "Golden retriever"



B "Cocker spaniel"



C "Dog"

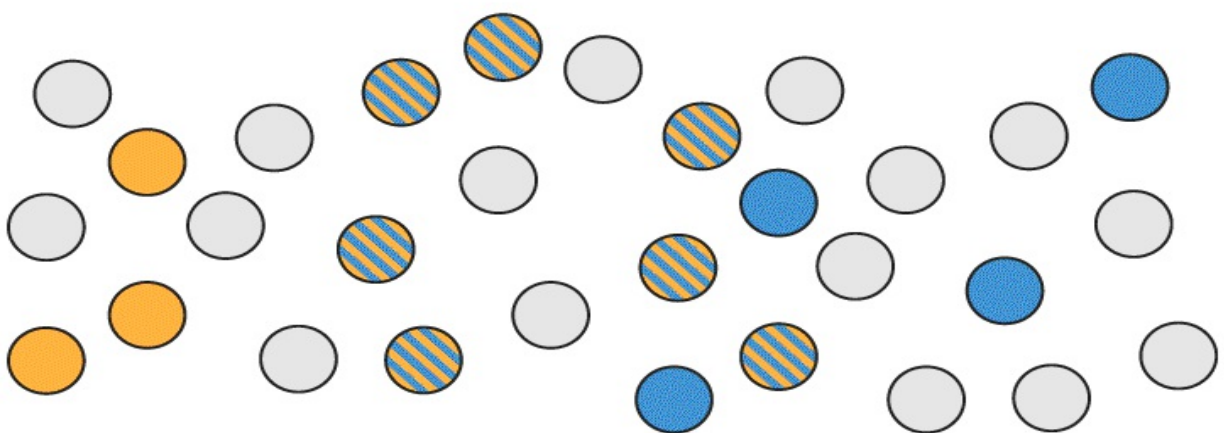


FIGURE 1.8 Distributed representations (A) Building on ideas from the earlier stimulus sampling theory of Estes, shown in [Figure 1.7](#), this distributed representation models “golden retriever” as activating a subset of nodes, shown in orange. (B) “Cocker spaniel” activates a different subset, shown in blue. (C) The similarity between them—both are dogs—emerges naturally as a function of the overlap between representations, shown by the orange-and-blue-striped nodes.

Description

The first illustration titled A Golden retriever shows a cluster, distributed across a pattern, with shaded and unshaded nodes. Ten nodes shaded represent Golden Retriever.

The second illustration titled B Cocker spaniel shows a cluster, distributed across a pattern, with shaded and unshaded nodes. Eleven nodes shaded represent Cocker Spaniel.

The third illustration titled C Dog shows a cluster, distributed across a pattern, with shaded nodes, unshaded nodes, and striped nodes. Three nodes shaded represent Golden Retriever, four nodes shaded represent Cocker Spaniel, and seven striped nodes represent the similarity between them.

connectionist models

Models that represent learning and other thought processes as networks of connections between simple processing units called nodes.

distributed representation

A representation in which information is coded as a pattern of activation distributed across many different nodes.

Connectionist models were inspired in part by ideas about how the brain is organized (see [Chapter 2](#)). Part of the expectation for connectionist models was that they would fulfill William James’s hope of arriving at a psychology that links brain physiology to behavior. Connectionist models laid the groundwork for a more complete integration of neuroscience and psychology, a goal that is also a core theme of this book.

After many productive years helping psychologists understand the computational power of networks of brain connections, David Rumelhart’s own brain networks began to fail him. In 1998, at the age of 56, he was diagnosed with Pick’s disease, an illness (similar to Alzheimer’s disease) that causes degeneration of the brain. For the last years of his life, until he died in 2011, he was no longer able to speak or recognize old friends and colleagues. Like Clive Wearing (see [“Learning and Memory in Everyday Life”](#)), David Rumelhart lost the vital memories that defined who he was.

Interim Summary

- Hermann Ebbinghaus was among the first to show that psychology, and especially the study

of human memory and forgetting, can be the subject of rigorous experimentation and quantitative analyses.

- Clark Hull attempted to devise formal mathematical models to describe learning.
- W. K. Estes, a student of B. F. Skinner who helped create the fields of mathematical psychology and cognitive science, built and tested mathematical models of learning that helped explain how random variation is as important to animal and human learning as it is to natural selection in evolution.
- George Miller adapted formal models of information theory to psychology to help us understand memory capacity.
- David Rumelhart revived William James's connectionist network models of memory but translated them into mathematical models of learning.

Synthesis

This chapter has introduced four fundamental themes that throughout history have driven the study of learning and memory. [Table 1.2](#) presents a brief recap of the chapter material, showing some of the ways the four themes have evolved and progressed over four successive eras in the history of this field of study: 400 BC–1880, with contributions from philosophy and natural history; 1880–1920, the birth of experimental psychology; 1920–1950, the reign of behaviorism; and 1950 to the present day, the birth of the [cognitive approach](#), which emphasizes higher mental activities in humans such as language, memory, and perception.

TABLE 1.2 Four fundamental themes and eras in the psychology of learning and memory

	Philosophy and natural history (400 BC–1880)	Birth of experimental psychology (1880–1920)	The reign of behaviorism (1920–1950)	The cognitive approach (1950–)
1.1 Linking Ideas in the Mind	<i>Aristotle (384–322 BC)</i> Memory depends on formation of associations. <i>William James (1842–1910)</i> Complex memories are networks of associations.	<i>Ivan Pavlov (1849–1936)</i> Animals learn to predict future events. <i>Edward Thorndike (1874–1949)</i> Behavior change depends on consequences.		
1.2 Nature Versus Nurture	<i>Plato (c. 427–347 BC)</i> Most knowledge is innate. <i>Aristotle (384–322 BC)</i> We are the sum of our experiences. <i>René Descartes (1596–1650)</i> Behaviors are innately determined reflexes. <i>John Locke (1632–1704)</i> The newborn mind is a blank slate (<i>tabula rasa</i>).		<i>John Watson (1878–1958)</i> Psychology should be limited to observable behaviors. <i>B. F. Skinner (1904–1990)</i> Free will is an illusion.	
1.3 Comparing Humans to Other Animals	<i>Aristotle (384–322 BC)</i> Animals remember but can’t recall. <i>Charles Darwin (1809–1882)</i> Inheritability, variation, and		<i>Edward Tolman (1886–1959)</i> Behavior reeks of purpose.	

	fitness = evolution.			
1.4 Formal Models and Universal Laws	<i>Aristotle (384–322 BC)</i> Contiguity, frequency, and similarity are the principles of association.	<i>Hermann Ebbinghaus (1850–1909)</i> Learning and forgetting can be studied rigorously.	<i>Clark Hull (1884–1952)</i> Comprehensive models can describe learning.	<i>William K. Estes (1919–2011)</i> Mathematical models describe learning processes. <i>George Miller (1920–2012)</i> Information theory explains short-term memory capacity. <i>David Rumelhart (1942–2011)</i> Distributed connectionist networks model memory like the brain.

cognitive approach

A subfield of psychology that emphasizes higher mental activities, such as language, memory, and perception.

What next? The past few decades have seen a revolution in the field (really fields) of learning and memory. And yet, as the field evolves, it continues to revolve around the four themes presented here. This is seen in [Chapter 2](#), which introduces some of the modern-day tools and approaches that are revealing the neural bases of the four essential themes. The remaining chapters supply further details that enrich our understanding of these key issues.

For example, [Chapter 2](#) describes how the work of Aristotle, James, Pavlov, and Thorndike on the psychological principles for linking ideas in the mind led to theoretical and experimental advances by Donald Hebb, Karl Lashley, and others who studied how brain systems for associative learning arise from biological processes. Aristotle’s three principles of associative learning—contiguity, frequency, and similarity—have each now been mapped onto specific neural processes in a mechanistic fashion and level of detail that Aristotle could never have imagined. In [Chapter 2](#) and other chapters, it becomes clear that current models of learning and memory in neuroscience, yielding exciting advances in our understanding of how specific brain circuits interact to produce learning and memory behavior, have built directly on James’s theories of complex networks of associations, Estes’s mathematical models of overlapping representations of stimuli, and Rumelhart’s computational models of memory in distributed networks.

The nature-versus-nurture debate continues to yield novel insights into mechanisms for learning and memory, including important discoveries in molecular biology that illuminate complex environmental influences modulating how genetics affects our brains and behavior. In turn, the efforts to understand cellular and molecular mechanisms for learning foster continued interest in the most elementary forms of learning in animals because these simple behaviors in the simplest animals have provided the best opportunity to dig deeply into the biological mechanisms that control learning and memory behavior. This endeavor and the growth of behavioral and cognitive neuroscience approaches to learning and memory have created new opportunities and needs for better understanding of the ways in which humans are similar to and different from other animals. Furthermore, our appreciation for the continuity of mechanisms across species, accompanied by advances in understanding molecular processes in worms and flies, has translated directly into important insights and, often, practical clinical applications for humans.

Chapter 1 Review

Know Your Key Terms

associationism

behaviorism

blind design

classical conditioning

cognitive approach

cognitive map

connectionist models

contiguity

data

dependent variable

distributed representation

double-blind design

dualism

empiricism

evolution

experimenter bias

extinction

forgetting

generalization

hypothesis

independent variable

latent learning

law of effect

learning

learning curve

mathematical psychology

memory

nativism

natural selection

operant conditioning

placebo

[psychology](#)
[radical behaviorism](#)
[reflex arc](#)
[response](#)
[retention curve](#)
[stimulus](#)
[subject bias](#)
[theory](#)

Quiz Yourself

1. According to Aristotle's principle of _____, we form a strong association between peanut butter and jelly because they appear together very often. ([p. 1](#))
2. Plato was a proponent of _____, which holds that the bulk of our knowledge is ingrained from birth. His student Aristotle was identified with a school of thought known as _____, which claims that our ideas stem from experience. ([p. 2](#))
3. The belief that the mind and body exist as separate entities is known as _____. A believer in this principle, _____ reasoned that a sensory stimulus and motor response follow a pathway known as the _____. ([p. 9](#))
4. According to Darwin, a trait can evolve through _____ if it is inheritable, variable, and _____. ([p. 16](#))
5. A contemporary of William James, _____ proposed that the psychology of memory can be defined precisely through mathematical laws as a rigorous natural science. ([p. 20](#))
6. How memory deteriorates over time is known as _____. Measuring how much information is retained at various intervals of time following learning, Ebbinghaus was able to plot a _____. ([p. 20](#))
7. Thorndike referred to the training in which organisms learn to make certain responses in order to obtain or avoid important consequences as _____, which is now

also known commonly as _____. (p. 8)

8. Emerging in the 1920s, the American approach to learning known as _____ centered around the argument that psychology should exclusively study observable behaviors. (p. 11)
9. Although the work of _____ is no longer considered relevant today, his students and followers carried on toward developing mathematical equations to explain learning. (p. 24)
10. In _____'s law of _____, behaviors that lead to desirable consequences are _____ likely to happen again in the future. (p. 8)
11. Edward Tolman, a neo-behaviorist, argued that his rats, intrinsically motivated to learn the general layout of mazes, had formed _____, internal psychological representations of the external world. (p. 17)
12. Learning that takes place even in the absence of any specific motivation to obtain or avoid important consequences is called _____. (p. 18)
13. The trial-by-trial variability in the elements we attend to when experiencing a stimulus is captured in the _____ proposed by _____. (pp. 25–26)
14. The number of digits in a standard phone number (not including area code) is relevant to the work of _____. (p. 27)
15. In the _____ models of cognition, ideas and concepts from the external world are not represented as distinct symbols but as patterns of activity over populations of many nodes. In these models, a _____ representation consists of the activation of many different nodes as opposed to only one. (pp. 28–29)

Concept Check

1. Categorize the following thinkers as taking either the empiricism approach or nativism approach: Aristotle, Plato, Descartes, Locke, James. In this regard, which earlier

thinkers influenced which later ones?

2. Several studies have shown what seems to be a genetic influence on some kinds of memory ability: parents with high memory ability are likely to have children who also have high memory ability. How would an empiricist account for such findings?
3. How did Tolman's latent learning studies challenge behaviorism? Could you develop an alternative interpretation of his studies that is consistent with behaviorism?
4. Give an example of a piece of information that consists of 3 bits and explain how you came up with it.

Answers to Test Your Knowledge

Who's Who in the History of Learning and Memory?

1. Aristotle; Descartes
2. Locke
3. James
4. Ebbinghaus; Pavlov; Thorndike

Borrowing from the Physical and Natural Sciences to Explain the Mind

1. How the body could function like a machine with input and output control pathways
2. How complex ideas could be formed from combinations of simpler and more elementary components
3. How psychology of memory could be a rigorous natural science, defined by precise mathematical laws
4. The distinction between a direct fixed connection and a modifiable indirect connection, as when a switchboard operator makes the call

5. That of all possible behavioral responses, the ones that are more successful and adaptive are more likely to be retained (i.e., learned)
6. The search for simple, powerful equations that unify many disparate observations
7. How to measure the amount of information in a message or stored memory, independent of the content

Chapter 2 The Neuroscience of Learning and Memory



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Structural Plasticity in Nervous Systems

[*Learning and Memory in Everyday Life: Top Five Tips for Faster Forgetting*](#)

[Brains](#)

[How Experience Changes Brain Structure](#)

Functional Properties of Learning and Memory Systems

[What Brains Do](#)

[How Remembering Changes Brain Activity](#)

Finding and Manipulating Memories

[Looking for Memories](#)

[How Researchers Change Brain Activity](#)

[*Learning and Memory in Everyday Life: Can a Pill Improve Your Memory?*](#)

IN THE MIDST OF A NEIGHBORHOOD BASEBALL GAME, a ball hit Orlando Serrell in the head so hard that it knocked him to the ground. At the time, he was only 10 years old, and like most boys who take a hit while playing with peers, he walked it off and eventually went back to the game. This seemingly innocuous incident proved anything but typical, however. Sometime after the hit, Orlando discovered an amazing ability: for any date after that fateful game, he could remember the day of the week on which that date fell, as well as what the weather was like on most of those days—without making any conscious effort to memorize these details or perform calculations with dates. Orlando's case is not unique but is an instance of a rare condition called *acquired savant syndrome* ([Trefft, 2014](#)). [Chapter 1](#) describes Clive Wearing, who lost many of his memory abilities after part of his brain was destroyed by a rare virus. Unlike Clive, individuals with acquired savant syndrome actually can *gain* prodigious memory capacities as the result of brain injury.

The startling implication of this phenomenon is that at least some (and perhaps all) human brains appear to have a much greater capacity for storing and recalling memories than people typically exhibit. If humans have hidden learning and memory capacities, might other animals also possess capacities of which we are currently unaware? If brains have such capacities, then why can't all individuals take full advantage of them? Might it be possible to develop neural technologies that improve a person's encoding and recollection of specific facts or erase memories of episodes one would prefer to forget?

The story of how scientists explore such questions, and identify the biological factors that

determine what an individual remembers or forgets, is the story of the neuroscience of learning and memory. Although scientists still have a long way to go in understanding how nervous systems work, they are compiling fascinating information about the brain's structure and functioning, as well as the ways in which it contributes to learning and memory. New imaging and sensing technologies allow researchers to observe healthy human brains as they form and retrieve memories, while new techniques for animal research allow researchers to measure and manipulate neural changes during learning. Insights into the neural mechanisms of learning and memory can help you understand how your actions may impact your own attempts to learn, remember, and in some cases forget the materials you study (for some ideas, see "[Learning and Memory in Everyday Life](#)").

LEARNING AND MEMORY IN EVERYDAY LIFE

Top Five Tips for Faster Forgetting

[Chapter 1](#) provides 10 tips for how you can change your behavior to improve your memory. In case you are more interested in erasing memories than retaining them (wasting time is fun!), you can also modify your brain function to improve your forgetting. Here is what you do.

1. *Don't sleep.* People who do not get enough sleep are less able to concentrate during the day, which makes it harder for them to encode new memories and retrieve old ones. Sleep is also important for transforming the learning of the day into long-term memories. Sleepy brains work worse. (More on this in [Chapter 7](#).)
2. *Stress out.* Stress generally interferes with recall. So, if you want to make retrieving memories particularly troublesome, just keep fixating on trying to remember things you can't, until frustration overcomes you. (More on this in [Chapter 7](#).)
3. *Overextend yourself.* The more things you try to keep in mind simultaneously, the greater the chance you will forget a bunch of them. So put aside all your note-taking devices—your pens, pads, computers, and iPhones—if you really want to maximize your loss. (More on this in [Chapter 9](#).)
4. *Deprive your senses.* The more impoverished your sensory inputs, the less likely you are to encode facts, events, and skills well enough to recall them later. Wear headphones, shades, and oven mitts. Minimize your brain activity. (More on this in [Chapter 3](#).)
5. *Be apathetic.* Nothing is more forgettable than something you couldn't care less about. Just keep chanting inside your head, "Whatever ... whatever ... whatever ...," and you can easily avoid the kinds of emotionally triggered brain states that make memories stick. (More on this in [Chapter 7](#).)

2.1 Structural Plasticity in Nervous Systems

Researchers in the field of **neuroscience**—the study of the brain and the rest of the nervous system—overwhelmingly believe that the brain is the seat of learning and memory. This was not always the prevailing opinion, however. When ancient Egyptians mummified a body, they first removed the organs they considered important and preserved them in special airtight jars—but they discarded the brain. Many centuries later, Aristotle, one of the most empirically oriented philosophers in history (whose ideas on memory are noted in [Chapter 1](#)), argued that the brain serves primarily to cool the blood. However, observations over the centuries since Aristotle’s time have convinced scientists that brain activity controls behavior and, by extension, the changes in behavior associated with learning and memory.

neuroscience

The study of the brain and the rest of the nervous system.

Historically, most early studies of learning and memory focused on observable behavior rather than on the brain and how it functions ([Chapter 1](#)). This is not because early learning and memory researchers were oblivious to the importance of the brain. Ivan Pavlov designed all of his behavioral experiments to answer questions about how the brain works. John Watson, the originator of behaviorism, started out studying how developmental changes in neural structures correlate with developmental changes in learning abilities. B. F. Skinner, perhaps the most famous behaviorist of the twentieth century, began his career as a physiologist. Why, then, did these researchers place so much emphasis on behavior and so little emphasis on the role of the brain?

Part of the answer is that brains are among the most complex structures in nature. Even as recently as 60 years ago, the complexity of the neural functions required for most learning tasks seemed incomprehensible. As new technologies became available, however, the study of brain function became more manageable. Today, aspects of brain function that previously were inaccessible are being measured daily in laboratories and medical institutions around the world. These new technologies have dramatically increased the number and productivity of studies exploring the neural substrates of learning and memory.

Brains

The brain is just one component—albeit a very important one—of a collection of body organs called the **nervous system**, the organ system devoted to the distribution and processing of signals that affect biological functions throughout the body. The tissues that are specialized for accomplishing these tasks include cells called **neurons**, which perform a variety of tasks, such as collecting incoming signals from sensory systems (leading to sight, taste, smell, touch, and sound) and from the rest of the body (indicating such conditions as hunger and sleepiness), processing these signals, and reacting to them by coordinating the body's responses (such as muscle movement and activity of internal organs).

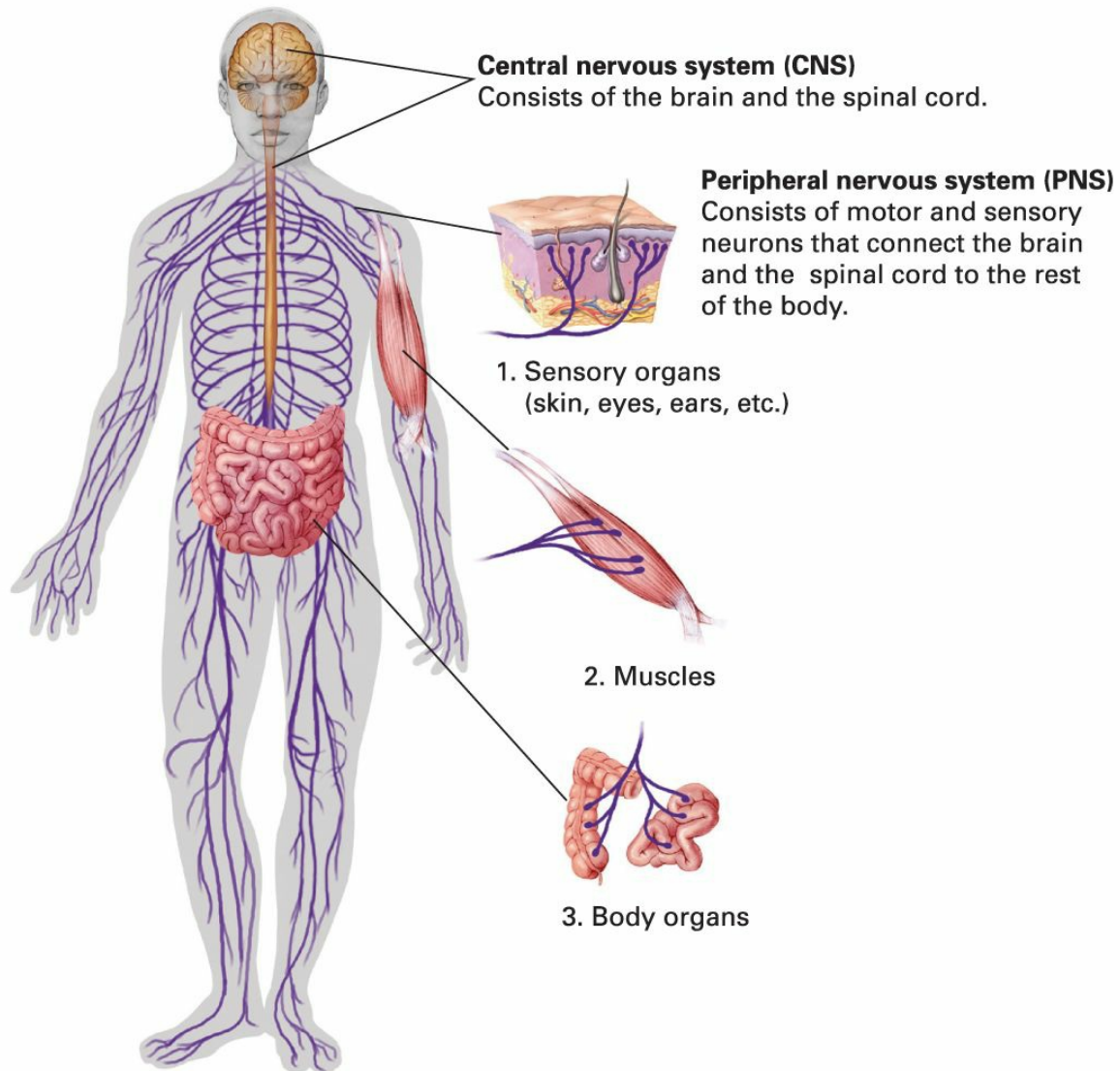
nervous system

An organism's system of tissues specialized for monitoring sensations, generating movements, and maintaining the function of most internal organs.

neuron

A special type of cell that is one of the main components of the nervous system.

In vertebrates, the nervous system can be divided into two parts: the central nervous system and the peripheral nervous system. The **central nervous system (CNS)**, where most of the events responsible for learning and memory take place, is made up of the brain and the spinal cord (**Figure 2.1**). The **peripheral nervous system (PNS)** consists of nerve fibers that connect sensory receptors (for example, visual receptors in the eye or touch receptors in the skin) to the CNS and of other fibers that carry signals from the CNS back out to the muscles and organs. Most of these fibers pass through the spinal cord, but a few—such as those from the light receptors in your eyes and those that activate the muscles controlling eye movements—travel directly to the brain without first making connections in the spinal cord.



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FIGURE 2.1 Nervous system components Every vertebrate has a central nervous system (CNS) and a peripheral nervous system (PNS). The CNS consists of the brain and spinal cord. The PNS consists of motor and sensory neurons that transmit signals between the CNS and the rest of the body: (1) sensory receptors in the skin, eyes, ears, and so on provide sensory inputs to the CNS; (2) motor fibers deliver directives from the CNS to muscles; (3) PNS fibers from the CNS regulate organs and glands.

Description

The parts are as follows. Central nervous system (CNS) comprising the brain and the spinal cord, Peripheral nervous system (PNS) comprising motor and sensory neurons that connect the brain and the spinal cord to the rest of the body: 1. Sensory organs (skin, eyes, ears, etc.), 2. Muscles, and 3. Body organs.

central nervous system (CNS)

The part of the vertebrate nervous system consisting of the brain and spinal cord.

peripheral nervous system (PNS)

The part of the nervous system that transmits signals from sensory receptors to the central nervous system and carries commands from the CNS to muscles.

Although every vertebrate possesses a CNS and PNS, there are big differences between the nervous systems of different species. Let's look at the vertebrate you are probably most familiar with: the human.

The Human Brain

The **cerebral cortex**, the tissue covering the top and sides of the brain in most vertebrates, is by far the largest structure of the human brain (**Figure 2.2A**). The word *cortex* is Latin for “bark” or “rind,” reflecting that the cortex, although about the size of the front page of a newspaper if spread out flat, is only about 2 millimeters thick. To fit inside the skull, the cerebral cortex is extensively folded, much like a piece of paper crumpled into a ball.

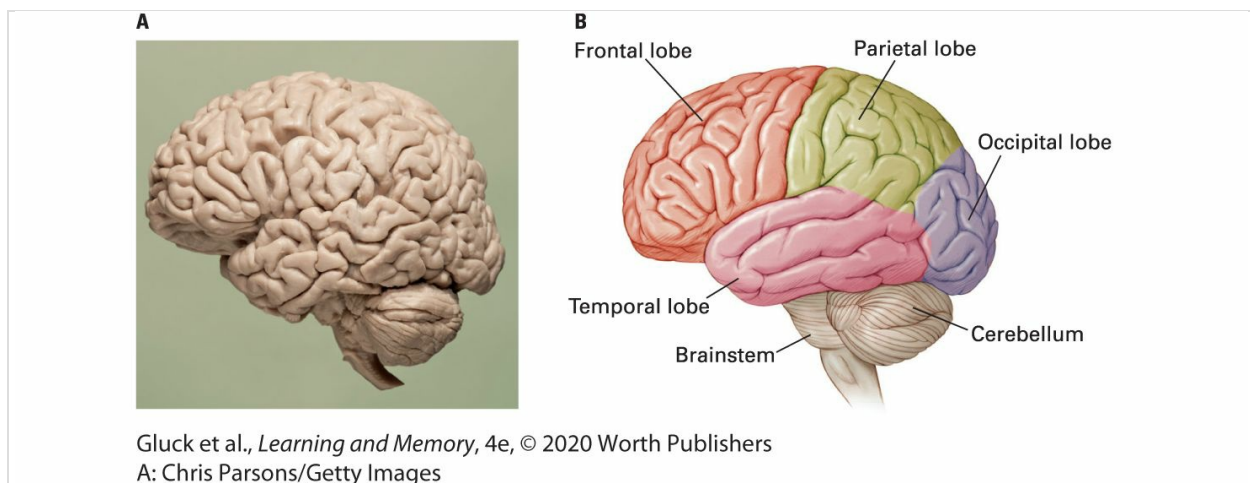


FIGURE 2.2 The visible surface of a human brain (A) A photograph of a human brain. (B) In each brain hemisphere, the visible cerebral cortex is divided into four principal areas: frontal lobe, parietal lobe, occipital lobe, and temporal lobe. Below the cerebral cortex are the cerebellum and brainstem. The brainstem connects the brain to the spinal cord.

Description

Section A shows the external view of the human brain.

Section B shows a labeled schematic of the human brain. The parts are as follows.

The cerebral cortex is divided into four major areas: frontal lobe, parietal lobe, occipital lobe, and temporal lobe. The anterior part of the brain is labeled, frontal lobe, it is the largest part of the brain. The adjacent part of the frontal lobe is labeled, parietal lobe, it is located at the top of the brain. The rearmost lobe of the cerebral hemisphere is labeled

Occipital lobe. It is located behind the parietal and temporal lobes. The second largest part of the brain is labeled, temporal lobes, it is located anterior to the occipital lobes and inferior to the parietal and frontal lobes. The cerebellum and brainstem are labeled inferior to the four lobes.

cerebral cortex

The brain tissue covering the top and sides of the brain in most vertebrates; involved in storage and processing of sensory inputs and motor outputs.

In humans, as in all vertebrates, the brain consists of two sides, or *hemispheres*, that are roughly mirror images of each other, so brain scientists, when talking about the cortex, may specify the “left hemisphere” or the “right hemisphere.” In each hemisphere, the cortex is divided further into the [frontal lobe](#) at the front of the head, the [parietal lobe](#) at the top of the head, the [temporal lobe](#) at the side of the head, and the [occipital lobe](#) at the back of the head ([Figure 2.2B](#)). The term *lobe* refers to the fact that these regions are anatomically distinct. The individual lobes got their somewhat odd names from the names of the skull bones that cover them. If you have trouble memorizing these four terms, remember: “Frontal is Front, Parietal is at the Peak, Temporal is behind the Temples, and the Occipital lobe is Out back.”

frontal lobe

The part of the cerebral cortex lying at the front of the human brain; enables a person to plan and perform actions.

parietal lobe

The part of the cerebral cortex lying at the top of the human brain; important for processing somatosensory (touch) inputs.

temporal lobe

The part of the cerebral cortex lying at the sides of the human brain; important for language and auditory processing and for learning new facts and forming new memories of events.

occipital lobe

The part of the cerebral cortex lying at the rear of the human brain; important for visual processing.

Subregions within each lobe are associated with a wide variety of perceptual and cognitive processes. For example, the frontal lobe helps you retrieve memories from your past, and enables you to keep track of multiple recent experiences; the occipital lobe allows you to see and recognize the world; the parietal lobe enables you to feel the differences between silk and sandpaper and to associate those differences with the look of silk and of sand; and the temporal lobe makes it possible for you to hear and to remember what you have done. We will discuss the functional roles of cortical subregions in greater detail throughout this book. Knowing the names and locations of the different lobes will help you keep track of what is happening where in your brain.

Sitting behind and slightly below the cerebral cortex is the **cerebellum** ([Figure 2.2B](#)), which contributes to the coordination of sensation and movements and is thus especially important for learning that involves physical action. At the base of the brain is the aptly named **brainstem** ([Figure 2.2B](#)), a collection of structures that connects the brain to the spinal cord and plays a key role in the regulation of automatic functions, such as breathing and regulating body temperature.

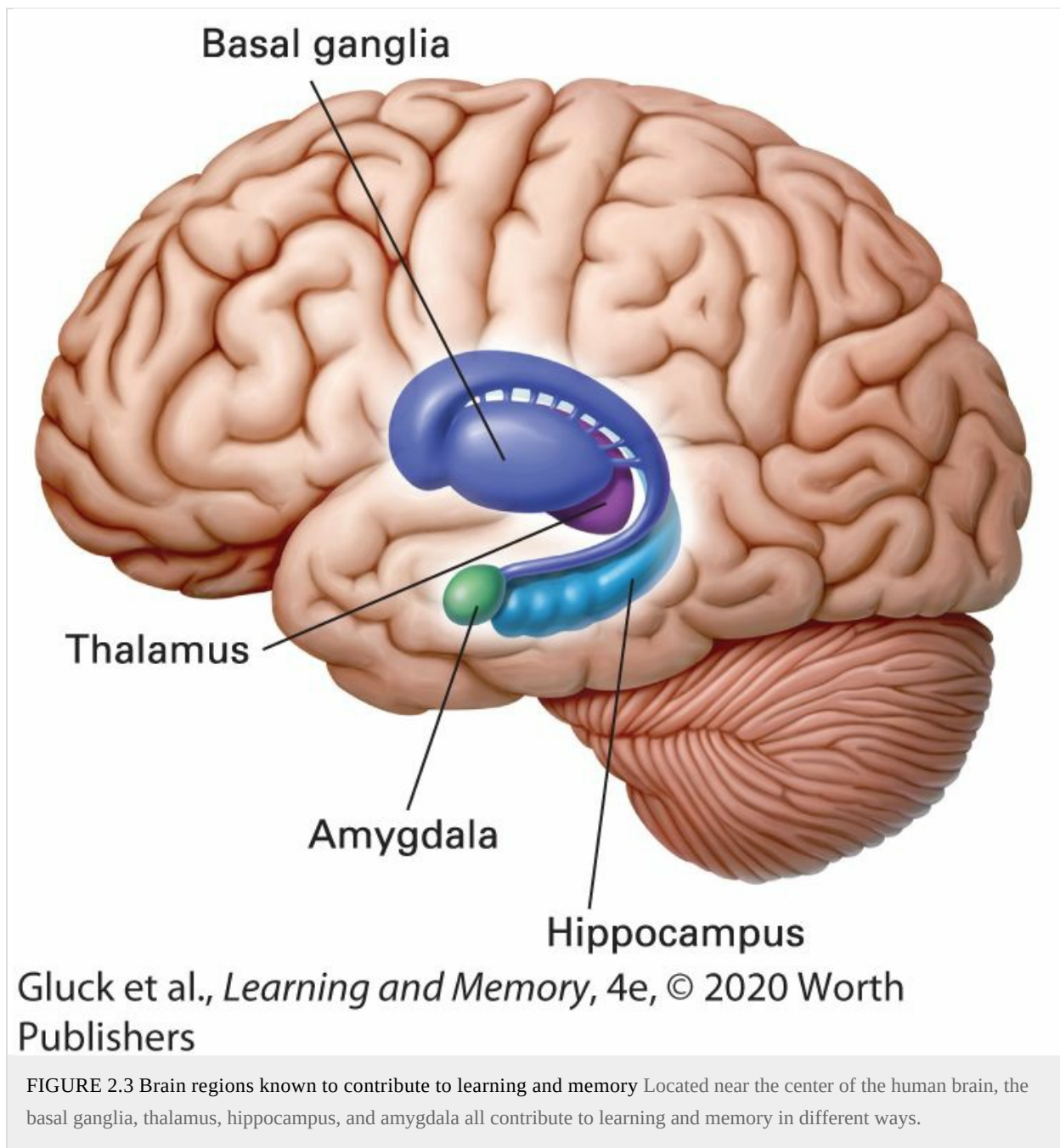
cerebellum

A brain region lying below the cerebral cortex in the back of the head. It is responsible for the regulation and coordination of complex voluntary muscular movement, including classical conditioning of motor-reflex responses.

brainstem

A group of structures that connects the rest of the brain to the spinal cord and plays key roles in regulating automatic functions such as breathing and body temperature.

Other brain structures, buried under the cerebral cortex, are not visible in photographs such as that shown in [Figure 2.2A](#). You will learn about many of these structures later in the book. For now, we will just introduce a few that are especially important for learning and memory ([Figure 2.3](#)).



Description

The labeled parts are as follows. Basal ganglia, Thalamus, Amygdala, and Hippocampus. The basal ganglia is located deep in the cerebral hemispheres of the brain. The Thalamus is located in the rostral end of the brainstem. The amygdala is located deep in the medial temporal lobe. It has an almond-shaped structure. The hippocampus is located in the temporal lobes of the brain. It has a horseshoe-shaped structure.

First, near the center of the brain lies the *thalamus*, a structure that receives various sensory

signals (associated with sight, sound, touch, and so forth) and connects to many cortical and subcortical regions. You can think of the thalamus as a gateway through which almost all sensory signals pass in order to affect brain activity. Sitting near the thalamus are the *basal ganglia*, a group of structures important for learning, planning, and producing skilled movements such as throwing a football or juggling. The *hippocampus* lies a little farther away, inside the temporal lobe; it is thought to be important for learning how to get places and for remembering autobiographical events (such as what you did last summer). Sitting at the tip of the hippocampus is a group of cells called the *amygdala*; this little brain region is important for emotional memories. If you remember the happiest—or saddest—day of your life, it is probably because your amygdala was particularly active at the time, adding emotional strength to those memories. Because you have two hemispheres, you actually have duplicates of each of these structures. For example, you have a left hippocampus and a right hippocampus and a left amygdala and a right amygdala.

Scientists are only beginning to understand what these brain areas do and how they relate to learning and memory, but it is becoming increasingly clear that it is a mistake to think of the brain as a single organ, like a liver or a kidney. Instead, the brain is a “society of experts,” with each region making its own specialized contribution to what we do and think.

Imaging Brain Structure

In the late 1800s, Franz Joseph Gall (1758–1828), a German anatomist and physiologist, pioneered the idea that different areas of the cerebral cortex are specialized for different functions. He also proposed that differences in character or ability are reflected in differences in the size of the corresponding parts of the cerebral cortex: that is, people with a special skill for languages would have a larger-than-average language area, and people with superior memories would have an overgrown memory area. Gall assumed that these differences in cortical size would be evident from bumps in a person’s skull. He developed a technique called [phrenology](#), in which he used skull measurements to predict an individual’s personality and abilities ([Gall & Spurzheim, 1810](#)).



“The prefrontal cortex is involved in higher mental functioning, like using a can opener and remembering to feed you.”

Mike Baldwin/Cartoonstock.com

phrenology

An obsolete field of study that attempted to determine mental abilities by measuring head shape and size.

Phrenology was quickly taken over by quacks who found various ways of making the idea pay. The main problem with phrenology, however, was that bumps on the skull are not caused by bulges in the underlying brain tissue. Gall did not know this because he had no way of examining the brain of a living person. Nearly two centuries later, technology finally advanced to the point where scientists could see inside the skulls of people who are still alive and begin to identify and measure brain structures that determine what individuals can learn and remember.

Modern techniques for creating pictures of anatomical structures within the brain are described collectively as [structural neuroimaging](#), brain imaging, or “brain scanning.” The images produced using these methods can show details of brain tissue and also brain [lesions](#), areas of damage caused by injury or illness.

structural neuroimaging

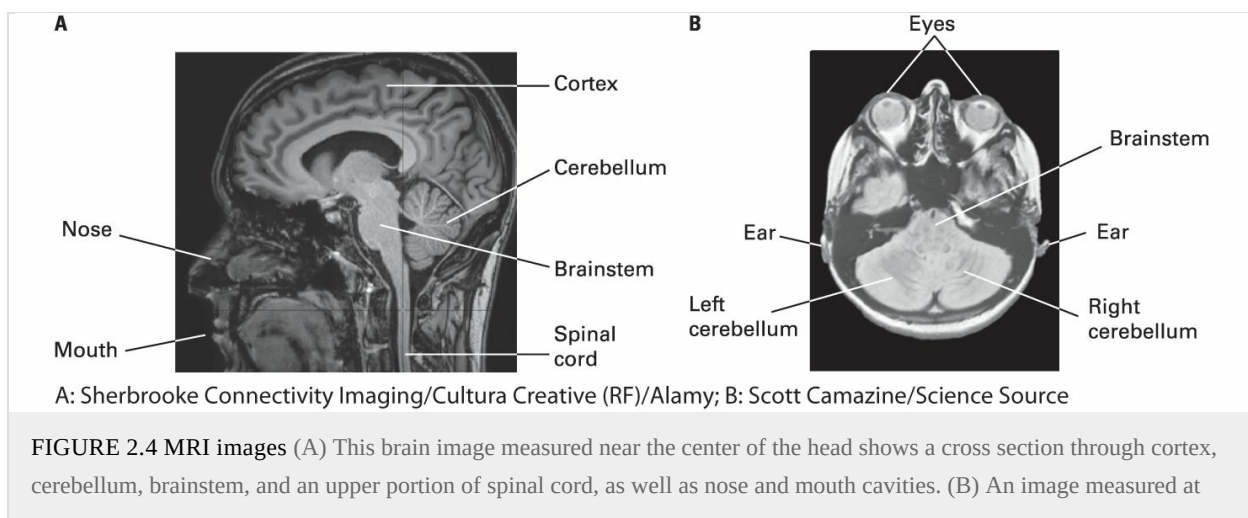
Techniques (such as MRI) for creating images of anatomical structures within the living brain.

lesion

Damage caused by injury or illness.

Structural neuroimaging provides a way not only to directly observe physical properties of a person's brain but also *to track changes in those properties over time*. These include changes that might occur as a function of aging, injury, disease, or learning experiences. Structural images of human brains are also critical for analyzing and interpreting changes in brain *function* that occur with learning, a topic we discuss in greater detail below.

Currently, brain images are most often produced through [magnetic resonance imaging \(MRI\)](#), in which changes in magnetic fields are used to generate images of internal structure. MRI employs an extremely powerful magnet, usually constructed like a giant tube. The person lies on a pallet that slides into the tube, and magnetic changes are induced in the brain tissues. The brain tissues are then allowed to return to normal. During this latter phase, a computer collects the different signals emitted by different tissues and uses them to generate images that look like photographs of a sliced brain. For example, [Figure 2.4A](#) shows an image comparable to what you would see if someone's head were sliced in half (minus the spewing blood), revealing a cross section of cerebral cortex, cerebellum, and brainstem, as well as some facial structures. An image measured at the level of the eyeballs, as in [Figure 2.4B](#), shows a different cross section. A different type of MRI, called [diffusion tensor imaging \(DTI\)](#), measures connections between brain regions, enabling researchers to study how major pathways between different brain regions change over time.



the level of the eyeballs (visible at the top of the image) contains little cortex (since the position is so far down in the person's head) but captures the low-hanging cerebellum.

Description

The imagery in section A shows a lateral sectional view of the human head with the following parts labeled: The cortex on the upper part of the brain, the cerebellum adjoining the brain stem, the brainstem at the center part of the brain connected to the spinal cord. The nose, mouth, and the spinal cord are also labeled.

The imagery in section B shows a sectional view of the human head, from the level of eyeballs. The parts labeled in section B are as follows. eyes, ears, brainstem, left cerebellum, and right cerebellum.

magnetic resonance imaging (MRI)

A method of structural neuroimaging based on recording changes in magnetic fields.

diffusion tensor imaging (DTI)

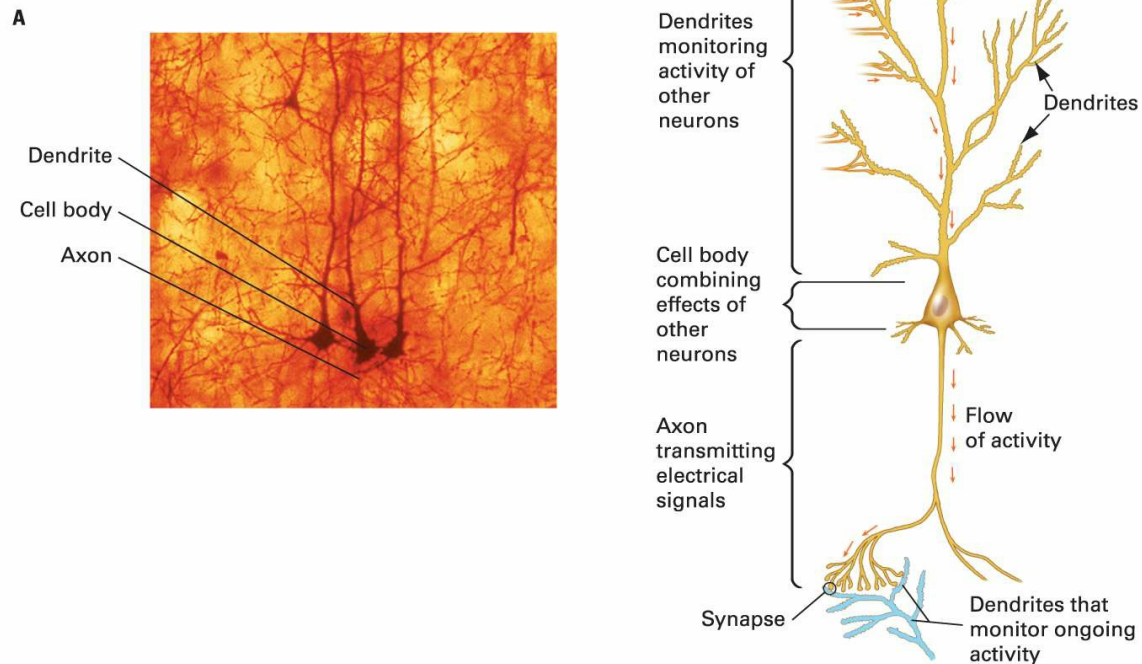
A type of MRI that measures connections between brain regions.

It is easy to confuse structural neuroimaging, used to visualize what brains are physically like, with a different kind of imaging, known as *functional neuroimaging*, which is described in [Section 2.2](#) and shows what brains are *doing* at the time of imaging. Both types of neuroimaging can reveal changes associated with learning, and examples of both are presented throughout this book. Whenever you come across a figure in which patches of color are superimposed on a picture of a brain, the first thing you should ask yourself is, do these colored regions show changes in brain structure (structural imaging), or do they show changes in brain activity (functional imaging)?

Neurons and Glia

Neurons are the building blocks of the nervous system. Some act as sensory receptors (such as those in the eyes, ears, and tongue that respond to visual, auditory, and taste stimuli), and some transmit signals from the spinal cord to the muscles. In vertebrates, many of the neurons are in the brain. Neurons are capable of changing their function and modifying the way they respond to incoming signals. These changes are thought to make learning and memory possible.

The prototypical neuron has three main components: (1) [dendrites](#), which are input areas that receive signals from other neurons; (2) the [cell body](#), or *soma*, which integrates signals from the dendrites; and (3) one or more [axons](#), which transmit signals to other neurons ([Figure 2.5](#)). For the most part, neural activity flows in one direction: from dendrites to axons.



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A: Biophoto Associates/Science Source

FIGURE 2.5 Neurons, the building blocks of brains (A) Brain tissue, stained to make neurons evident and photographed through a powerful microscope. The pyramid-shaped cell bodies and interconnecting branches of several neurons are visible. (B) The prototypical neuron has three main components: dendrites for monitoring the activity of other neurons, a cell body (soma) that integrates incoming signals, and one or more axons that transmit signals to other neurons. Neural activity flows mainly from dendrites to axon(s).

Description

The micrograph shows a network of neurons with the cell body, thread-like dendrites and axons labeled. Dendrites and axons are at the opposite ends of the cell body.

The schematic on the right shows the structure of a neuron. The flow of activity is from the top dendrites, down through the cell body, and out the axons at the bottom. Text corresponding to different parts of the neuron read as follows.

1. Dendrites: synapses with axons from other neurons that modulate activity are labeled. Dendrites monitor activity of other neurons.
2. Cell body: combines effects of other neurons.
3. Axons: transmit electrical signals to dendrites that monitor ongoing activity. A synapse is labeled at the end of a

dendrite at the bottom of the neuron.

dendrite

On a neuron, an extension that is specialized to receive signals from other neurons.

cell body

The central part of the prototypical neuron; it contains the nucleus and integrates signals from all the dendrites. Also known as the *soma*.

axon

The output extension of a neuron, specialized for transmitting signals to other neurons or to muscles.

When the incoming signals arriving at the cell body have a total electrical charge that exceeds a certain threshold, the neuron “fires,” propagating an electrical charge called an *action potential* down its axon. This propagation is an all-or-nothing event: either the neuron fires or it does not; there is no in-between stage.

It is convenient to talk about a “prototypical neuron.” But in reality, neurons, like brains, come in a wide array of shapes and sizes. For example, *pyramidal cells* are neurons with pyramid-shaped cell bodies (shown in [Figure 2.5A](#)); *stellate cells* have star-shaped cell bodies. A neuron may have a single axon, two axons, or many axons. Neurons known as *interneurons*, which connect two or more neurons, have short axons or no axons at all. The neurons that carry signals from the spinal cord to the feet have axons that stretch a meter or more in humans. The various shapes and sizes of different neurons undoubtedly contribute to their function. But, in many cases, neuroscientists do not know the specific advantages that a particular shape or size provides.

Neurons are not the only kind of cell in the brain; they are matched in number by various species of cells known as [glia](#). *Astrocytes* are glia that line the outer surface of blood vessels in the brain and may help in the transfer of oxygen and nutrients from the blood to neurons. Glia called *oligodendrocytes* wrap the axons of nearby neurons in *myelin*, a fatty substance that insulates electrical signals transmitted by neurons, thus speeding the transmission of signals down the axon. Glia are as important as neurons for normal brain (and overall central nervous system) function. For example, multiple sclerosis is a disease in which the myelin coating of axons degenerates; this interferes with neural function, leading to jerky muscle movements and impaired coordination, as well as problems with vision and speech.

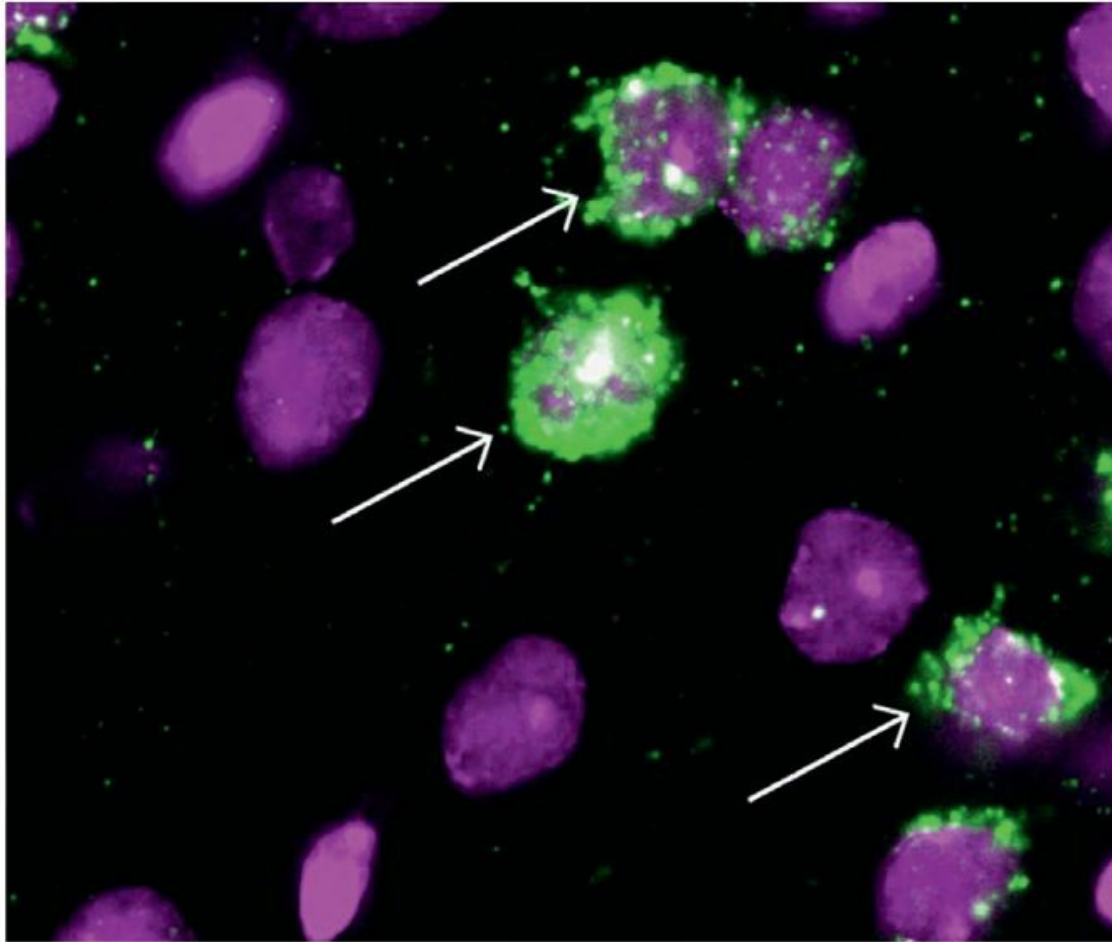
glia

Cells of various types that provide functional or structural support to neurons; some contribute to changes in connections between neurons.

Traditionally, researchers thought that glia mainly provide functional and structural support to neurons. New research shows, however, that glia directly contribute to learning and memory by actively controlling changes in connections between neurons ([Alberini et al., 2018](#); [Fields et al., 2014](#)). Even so, most neuroscientists who study the neural bases of learning and memory focus their efforts on understanding neurons: how they control behavior and how they change during learning.

How Experience Changes Brain Structure

[Chapter 1](#) describes some early theories of *associationism*, in which memory depends on forming links between experienced events, and of *conditioning*, in which repeated pairing of stimuli or responses with behaviorally relevant events brings about changes in behavior. [William James \(1890\)](#) proposed that the links created during learning and memory formation are not just conceptual connections but actual, physical properties of brains. According to James, the ability to learn and remember shows that brain structure can be changed by experience. He believed that brain structures gradually change when activity repeatedly flows through specific pathways; like Ivan Pavlov, James thought that it is this [plasticity](#) of brain tissue that makes learning possible.



Sakaguchi & Hayashi, 2012. Under the terms of the Creative Commons Attribution 2.0.

Techniques for imaging neurons in brains make it possible to visualize structural changes that occur during learning. Do you think the changes shown here in green occurred in the dendrites, soma, or axons of these neurons?

plasticity

The capacity of brain structure to change over time.

There are many ways in which experience might change the brain. Networks of neurons can be rewired or replaced. Functional features of neurons might be strengthened or weakened. New functions or even new neurons might be introduced. In the case of learning and memory formation, there is evidence that all these kinds of structural plasticity and more occur.

Imprinting

Much of what is known about the neural bases of learning and memory comes from studies of

animals other than humans. Many aspects of a mouse brain, a monkey brain, a bird brain, and even an insect brain are similar enough to a human brain to have made this possible (as predicted by Darwin's theory of natural selection, described in [Chapter 1](#)). Comparative studies provide a foundation for understanding how structural changes in brains relate to learning and memory abilities.

One unique case of learning that has proven particularly informative is *imprinting*, in which a newborn animal forms a lifelong attachment to whatever movements it views early on—typically, those of the animal's mother. Imprinting is more complicated than it might seem at first. It involves elements of *classical conditioning*, *perceptual learning* (discussed in [Chapter 3](#)), recognition memory (also discussed in [Chapter 3](#)), and *operant conditioning* ([McCabe, 2013](#)). In addition, imprinting occurs only within a short window of time after birth. We will return to the topic of imprinting when we discuss developmental aspects of learning and memory ([Chapter 12](#)). For now, you just need to know that the memory produced by imprinting is one of the first memories some young animals form and that it is specific enough to enable youngsters to spot their mother in a lineup.

Special chemical solutions have been developed which make it possible to locate neurons that have changed as a function of specific learning experiences. Usually, the brain tissue has to be bathed in the chemicals, and the dyed neurons are visible only through a microscope, so the brain must be removed from the animal soon after the learning occurs. Using such methods, researchers identified a specific area in young chicks' brains that showed extensive structural changes after the chicks imprinted on visual stimuli ([Horn, 2004](#)). As Gall's ideas about division of labor in the brain suggest, there appears to be a specialized “mother memory module” that enables chicks to rapidly learn what their mothers look like. Neuroscientists have discovered that neurons in this region change in many ways, depending on the specific experiences chicks have, how much the chicks learn from those experiences, how well the chicks sleep afterward, and whether the neurons are located in the left or right side of the brain ([McCabe, 2013](#)). Learning who your mom is can require a bit of brain rewiring if you are a bird.

Environmental Enrichment

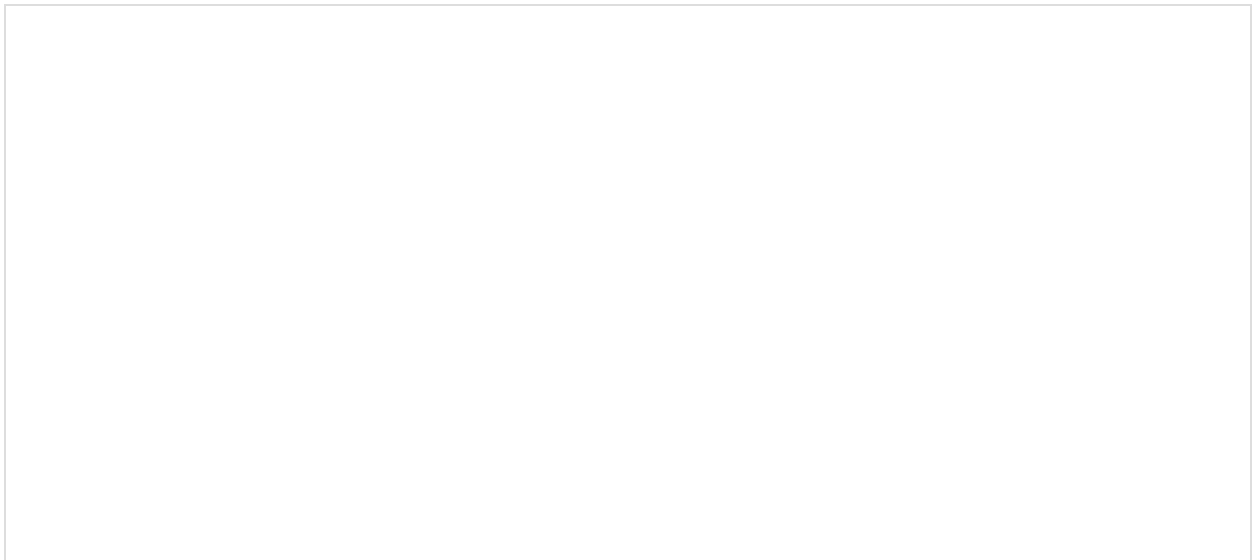
Early studies of brain structure in rats found that simply providing young rats with more opportunities for learning, social interactions, and exercise could lead to visible changes in their

neurons. Researchers housed one group of rats in an [enriched environment](#), where there was plenty of sensory stimulation and opportunity to explore and learn. For the rats, this meant a large cage filled with toys to play with and other rats with whom to socialize. A second group of rats—the *control*, or comparison, group—lived in standard laboratory housing, each rat isolated in a small chamber that contained nothing but a drinking spout and food cup. The results? The rats housed in the enriched environment showed better maze learning than the rats kept in standard laboratory housing ([Renner & Rosenzweig, 1987](#); [Rosenzweig, 1984](#)), and as mentioned, these increased learning capacities were associated with structural changes in their neurons.

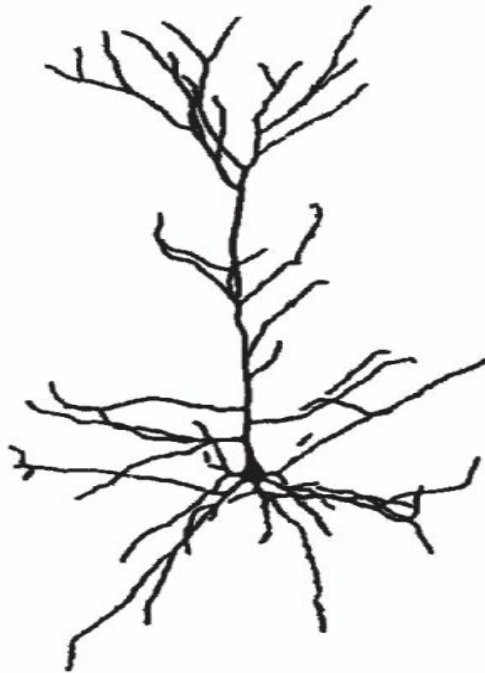
enriched environment

An environment that provides sensory stimulation and opportunities to explore and learn; for a rat, this may mean housing in a large cage with many toys to play with and other rats to socialize with.

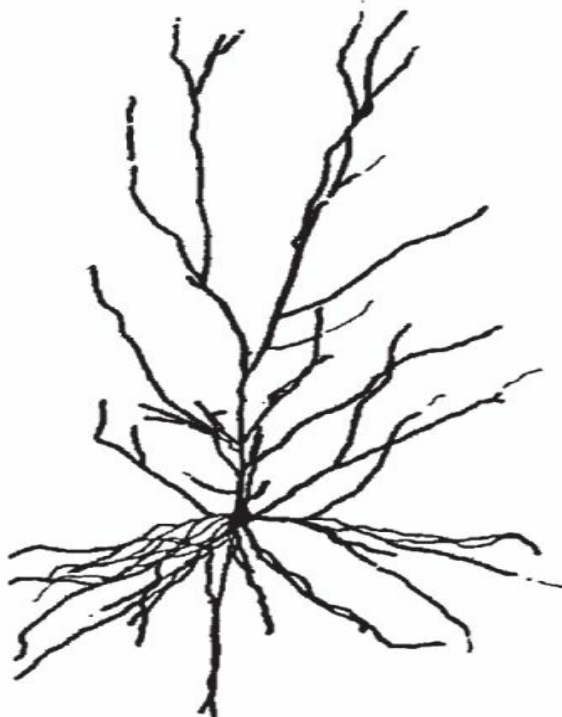
Rats raised in an enriched environment have cortical neurons with more and longer dendrites than experience-impooverished control groups ([Figure 2.6](#)). The dendrites of rats in the enriched environment also have more connections with other neurons ([Globus, Rosenzweig, Bennet, & Diamond, 1973](#); [Greenough, West, & DeVoogd, 1978](#)). These neural changes occur quickly: as few as 60 days of housing in an enriched environment can result in a 7 to 10% increase in brain weight of young rats and a 20% increase in the number of connections in the visual cortex. Similar changes are seen in the brains of monkeys and cats raised in enriched environments. Even the brains of fruit flies housed in large communal cages with visual and odor cues show similar changes, compared with flies housed alone in small plastic vials ([Technau, 1984](#)).



A Standard laboratory housing



B Enriched laboratory environment



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FIGURE 2.6 Deprived environment versus enriched environment Representations of neurons from the cortex of (A) a rat raised in standard laboratory housing and (B) a rat raised in an enriched laboratory environment. Neurons from rats raised in enriched environments typically have more and longer dendrites than experience-impooverished controls.

Description

The first schematic shows a neuron in a standard laboratory housing, with less dendritic growth. The second schematic shows a neuron in an enriched laboratory environment with greater dendritic growth.

Do similar effects occur in humans? Preschool children placed in “high-quality” day care (with lots of toys, educational experiences, and teacher interaction) often fare better in elementary school than children whose day care offers fewer opportunities for learning ([Peisner-Feinberg, Burchinal, & Clifford, 2001](#)). There isn’t yet definitive evidence that human brains undergo enlargement similar to that of rats after environmental enrichment because the current structural neuroimaging approaches used on children do not have the resolution necessary to detect changes in individual neurons. However, suggestive data come from a study of London taxi drivers.

On-the-Job Training

London is a sprawling city with hundreds of small, crooked streets. To receive an official license, London taxi drivers must study for up to 3 years and pass a grueling exam that, for example, requires them to indicate the shortest path between random London addresses. This means that licensed London taxi drivers are a group of people sharing an extensive fund of spatial knowledge.

Researcher Eleanor Maguire and her colleagues used MRI to compare brain volumes in a group of London taxi drivers with those of age-matched Londoners who had not studied the geography of their city so extensively ([Maguire et al., 2000](#)). The only part of the brain that differed significantly between the groups was the hippocampus: the taxi drivers had slightly larger hippocampal volumes than non-taxi drivers. Furthermore, the size of the hippocampus differed even among individual taxi drivers: those who had been driving for more than a decade had a larger volume than those who had been driving for only a few years. One possible interpretation of these volume differences is that the intensive spatial learning in taxi drivers causes an increase in dendritic branching in hippocampal neurons—making those neurons take up more room, just

like the rat neurons shown in [Figure 2.6](#).

Hebbian Learning

Any physical change in neurons, or in the systems that support them (such as glia and blood vessels), can affect **neural connectivity**—the connections between neurons through which brain systems interact. The idea that connections between neurons change during learning was first popularized by Santiago Ramón y Cajal (1852–1934), a Spanish physiologist and anatomist whose work in neuroanatomy won him the Nobel Prize in Physiology or Medicine in 1906. Specifically, Ramón y Cajal theorized that learning involves strengthening or weakening connections between individual neurons ([Ramón y Cajal, 1990 \[1894\]](#)). This same basic idea was also proposed by William James, who, as mentioned earlier, believed that changes in physical connections within the brain determine how memories are linked together.

neural connectivity

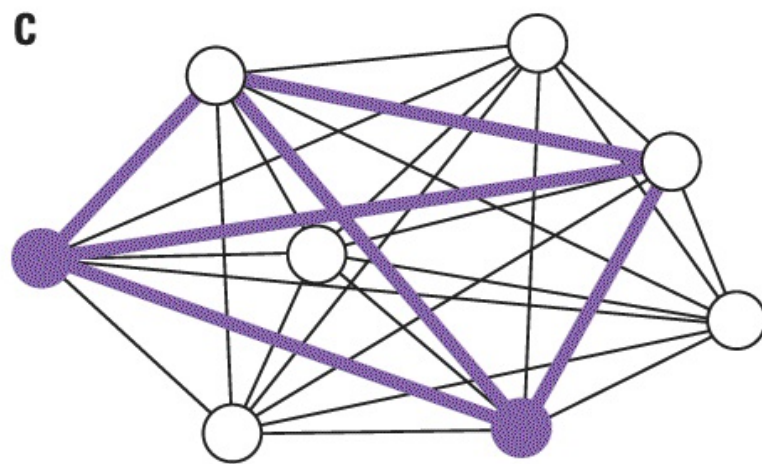
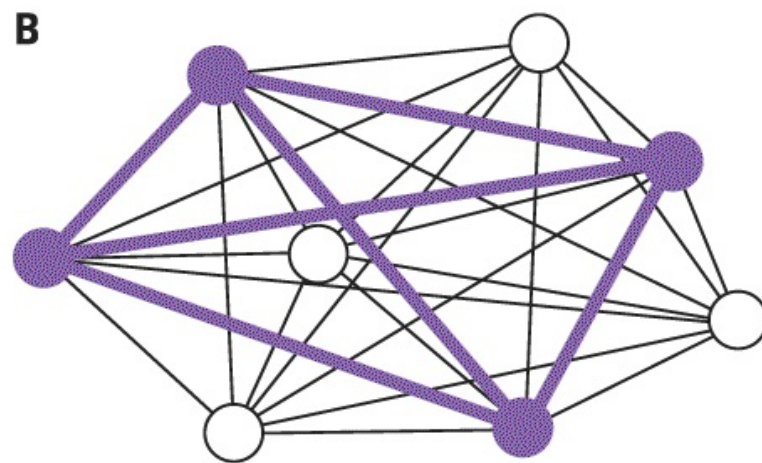
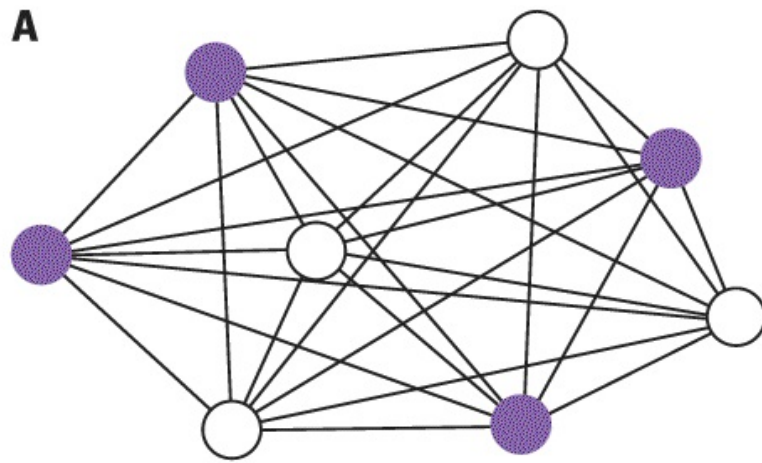
The connections between neurons through which brain systems interact.

But how exactly can the right connections between neurons be weakened or strengthened by learning experiences? One of neuroscience’s enduring insights regarding the neural substrates of learning came from Donald Hebb, a Canadian neuroscientist. In one of the most often quoted passages in neuroscience, Hebb wrote, “When an axon of cell A is near enough to excite a cell B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place such that A’s efficiency, as one of the cells firing B, is increased” ([Hebb, 1949, p. 62](#)). In other words, if two neurons—we will call them neuron A and neuron B—are connected, and if they often fire at nearly the same time, then the connections between them should be strengthened, “wiring” the two neurons together. This would increase the probability that whenever neuron A became active, it would cause neuron B to become active, too. A shorthand version of this “rule” that neuroscientists often use is *neurons that fire together, wire together*.

Learning that involves strengthening connections between neurons that work together is called **Hebbian learning**. [Figure 2.7](#) shows a simple model of Hebbian learning. Eight hypothetical cortical neurons are shown, each with weak connections to surrounding neurons ([Figure 2.7A](#)). Now let’s assume that some sensory stimulus evokes activation in a subset of these neurons that are tuned to features of this stimulus (solid circles in [Figure 2.7A](#)). As those neurons become active, they produce outputs that are transmitted to other nearby neurons. According to Hebb’s

rule—neurons that fire together wire together—the connections between coactive neurons are strengthened as a result. Repeated coactivity of the same subset of neurons, in response to the same stimulus, has a cumulative effect, resulting in the strong connections (heavy lines) shown in [Figure 2.7B](#). Thus, repeated exposure to a stimulus can strengthen connections within a distinctive subset of cortical neurons; this subset can then provide an increasingly reliable basis for identifying the stimulus that is activating them. Changing the connections between cortical neurons creates a pattern that makes a repeated stimulus more likely to be recognized and distinguished from other stimuli.





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FIGURE 2.7 A simple model of Hebbian learning Circles correspond to cortical neurons, and lines denote connections between them. (A) Stimulus inputs activate a subset of the neurons (solid circles). (B) Connections between coactive neurons are strengthened (heavy lines). (C) After connections between coactive neurons have been established, an incomplete version of a familiar stimulus may activate just some of the neurons (solid circles) in the subset that represents the stimulus. Activation flows along the strengthened connections and ultimately retrieves the complete stimulus, resulting in the representation shown in part B.

Description

The first illustration shows stimulus inputs activating a subset of the neurons, represented by 4 solid circles and 4 empty circles connected through lines.

The second illustration shows a strengthened connection between coactive neurons. All the lines between the solid circles become heavy lines.

The third illustration shows 2 solid circles and 2 empty circles. The two solid circles are connected with two empty circles through heavy lines.

Hebbian learning

The principle that learning involves strengthening the connections of coactive neurons; often stated as, “Neurons that fire together, wire together.”

Hebbian learning also explains how repeated experiences can enhance the ability to recognize familiar stimuli (discussed further in [Chapter 3](#)). Suppose that once connections have been established between cortical neurons, the organism encounters an incomplete version of a familiar stimulus ([Figure 2.7C](#)). Only some of the neurons that represent that familiar stimulus are activated at first (solid circles in [Figure 2.7C](#)), but the connections already established through repeated experiences will produce outputs that complete the familiar pattern, reconstructing [Figure 2.7B](#). Similarly, recognition of distorted versions of a familiar stimulus, such as might occur when you meet an old friend who has dyed her hair, could also be facilitated by stored patterns encoded as connections between neurons that on previous occasions were simultaneously active.

According to Hebb, learning-related changes in connections between neurons are an automatic result of the neurons’ mutual activity and the brain’s capacity for structural plasticity. We now know that Hebb was on the right track. But it was several more decades before technology advanced to the point where researchers could directly monitor such experience-related changes in neural activity. The ability to observe functional changes in brains as individuals learn and perform memory-based tasks has led to a host of new discoveries about how learning and

memory work, samples of which we review throughout this book.

Interim Summary

- The brain and spinal cord make up the vertebrate central nervous system (CNS). The brain controls behavior through connections with the peripheral nervous system (PNS), which consists of sensory neurons coming from sensory receptors and motor neurons going to body muscles.
- The vertebrate brain is made up of several different regions that contribute to learning and memory, including the cerebral cortex, cerebellum, hippocampus, basal ganglia, and amygdala.
- Neurons, the building blocks of the nervous system, are capable of changing their function and modifying the way they process inputs.
- Modern structural brain-imaging techniques (including MRI and DTI) provide ways to measure variations in the brain structure of living humans without causing harm.
- Techniques for imaging neural structures make it possible to collect detailed information about neural changes that occur during learning.
- Enriched environment studies show that learning experiences can have a profound impact on brain structure, as well as on an individual's learning and memory abilities.
- The ability of brains to change with experience is called *structural plasticity*. Strengthening or weakening the connections between neurons is thought to be a primary mechanism of memory formation.

2.2 Functional Properties of Learning and Memory Systems

Modern brain scientists assume that brains are composed of multiple systems that specialize in collecting, processing, and storing particular kinds of information. But there is not always the one-to-one relationship supposed by Gall and other phrenologists, in which each individual function or ability is performed in a dedicated corner of the brain. Instead, one brain area may play a role in many functions, and one function may rely on contributions from many brain areas.

What determines how brain regions contribute to learning and memory processes? Two major factors are the kinds of *input* a region receives and the kinds of *output* it produces. These inputs and outputs are closely related to the stimuli and responses that behaviorists emphasized in their theories of learning (reviewed in [Chapter 1](#)).

What Brains Do

[Chapter 1](#) defines *learning* as a process by which changes in behavior arise as a result of experience. Thus, when Pavlov's dogs began to salivate after hearing a sound that predicted food, this change in behavior—salivation in response to a sound—provided evidence that the dogs had learned about the relationship between the sound and the food. But even before Pavlov began using the dogs in his experiments, they would salivate in response to food. Salivation during eating is a reflexive behavior that dogs (and other mammals) develop early in life; it helps the digestive system get ready to process incoming food.

A [reflex](#) is an involuntary and automatic response that is “hardwired” into an organism; in other words, it is present in all normal members of a given species and does not have to be learned. Just like Pavlov's dogs, humans salivate when eating food. This is only one of several reflexes that humans are biologically prepared to perform: newborns suck when they encounter a nipple (sucking reflex), hold their breath when submerged underwater (the diving reflex), and grasp a finger so tightly that they can support their own weight by hanging on to it (the palmar grasp reflex). Adults have reflexes, too, such as the knee-jerk reflex when the doctor hits your knee with a rubber mallet and the eyeblink reflex when someone blows air at your eye.



Elli Thor Magnusson/Getty Images

Why isn't this infant drowning?

reflex

An involuntary and automatic (unlearned) response.

[Chapter 1](#) describes René Descartes and his belief that reflexes are hydraulic movements caused by spirits flowing from the brain into the muscles. For many years, scientists accepted this explanation, assuming that there must be some kind of fluid carrying instructions from the brain to the muscles. It wasn't until the early twentieth century that researchers discovered, first, that there is no such fluid and, second, that the brain is not in absolute control of the muscles at all.

Instead of a hydraulic fluid, there are two distinct types of nerve fibers (bundles of axons) connecting the muscles to the spinal cord—one set of fibers carrying sensory signals from the peripheral nervous system into the spinal cord, and a second set carrying motor signals back from the spinal cord to the muscles ([Bell, 1811](#); [Magendie, 1822](#)). If a pinprick or other painful stimulus is applied to a dog's leg, its leg jerks reflexively (just as you would automatically pull your leg away if someone pricked you). If the sensory fibers are cut, the dog's sensation of pain disappears, and the reflex fails to occur—although the dog can still move its leg voluntarily. On the other hand, if the motor fibers are cut, the animal can still feel pain but again does not make reflexive leg movements (or voluntary ones). From such observations, scientists deduced that in the spinal cord, sensory fibers are separate from motor fibers. They run in two parallel nerve pathways: one devoted to sensing and the other to responding. This finding, called the *Bell–Magendie law of neural specialization*, represents the historical first step toward understanding the neural mechanisms of learning. Specifically, it shed light on how the nervous system responds to stimuli and how it controls responses evoked by those stimuli.

Following up on the discovery of neural specialization, English physiologist Charles Sherrington (1857–1952) conducted many studies on dogs whose spinal cords had been surgically disconnected from their brains so that the spinal cord no longer received any brain signals. Such surgically altered dogs show many basic reflexes, such as jerking their leg away from a painful stimulus. Because the brain cannot contribute to these reflexes, they must be generated by the spinal cord alone. In fact, we now know that sensory inputs can activate motor fibers traveling out of the spinal cord, without requiring signals from the brain. (The sensory pathways in the spinal cord *are* largely separate from the motor pathways there, as noted above; yet at the same time, sensory and motor neurons are closely interconnected throughout the nervous system.) If you have ever stuck your hand into dangerously hot or cold water and jerked it away almost

before realizing what you have done, or watched your knee jerk in response to the tap of a doctor's rubber mallet, then you have experienced your spinal cord responding without receiving any help from your brain.

Sherrington concluded that such simple “spinal reflexes” can be combined into complex sequences of movements, and these reflexes are the building blocks of all behavior ([Sherrington, 1906](#)). Sherrington's description of reflexes differed from that of Descartes in assuming that spinal reflexes did not depend on the brain and did not involve the pumping of spirits or fluids into the muscles. Sherrington received a Nobel Prize in 1932 for his work in this area, and he is now considered to be one of the founding fathers of neuroscience. His ideas provided the groundwork and motivation for Pavlov's early investigations of reflex conditioning in dogs ([Pavlov, 1927](#)) and have continued to influence learning and memory researchers ever since.

If the spinal cord controls reflexes and if complex actions can be described as combinations of these reflexes, then where does the brain come in? Sensory fibers enter the spinal cord and connect to motor fibers there, but some fibers also travel up to the brain. The brain processes these inputs and produces its own outputs, some of which may travel back down the spinal cord and out to the muscles. The parallel sensory and motor pathways traveling up and down the spinal cord to and from the brain are similar to the parallel sensory and motor pathways that were identified traveling into and out of the spinal cord.



“Level with me, guys. Do any of you really understand what I’m saying, or is this just Male Pride Nodding Reflex?”

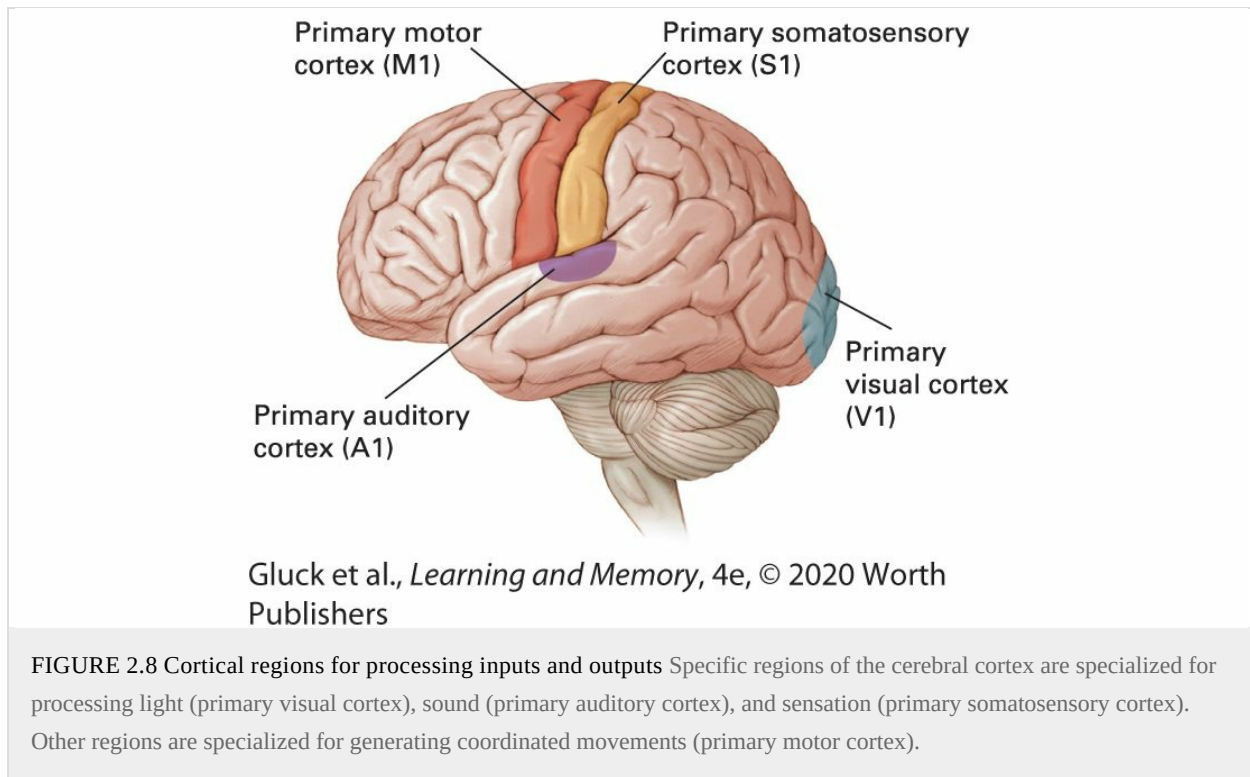
Bradford Veley/Cartoonstock.com

Traditionally, reflexes are presumed to function the same way in men and in women. But what kinds of reflexes might be sex specific? ([Chapter 12](#) considers how genetic differences between sexes can affect learning abilities.)

Incoming Stimuli: Sensory Pathways into the Brain

We’ll focus first on the sensory pathways that provide inputs to the brain. As noted in [Section 2.1](#), most sensory inputs enter the brain through the thalamus. The thalamus in turn distributes these inputs to cortical regions specialized for processing particular sensory stimuli, such as the primary auditory cortex (A1), for sound; the primary somatosensory cortex (S1), for sensations from skin and internal organs (such as touch and pain); and the primary visual cortex (V1), for sight. A1 is located in the temporal lobe, S1 in the parietal lobe, and V1 in the occipital lobe ([Figure 2.8](#)). Such areas are collectively called *primary sensory cortices*, as they are involved in the first stage of cortical processing for each type of sensation. Each primary sensory cortex can

then transmit outputs to surrounding cortical regions for further processing. For example, the primary visual cortex may start the processing of stimuli from the eye by extracting simple features—say, lines and shading—from a visual scene; later stages of cortical processing elaborate by detecting motion or shape in the scene and, finally, by responding to features of individual objects and their meaning. Damage to primary sensory cortices can eliminate particular perceptual abilities. For instance, people with damage to V1 can become blind, even though their eyes are in perfect working order, and damage to A1 can cause deafness.



Description

Primary motor cortex (M1), the primary somatosensory cortex (S1), primary auditory cortex (A) and Primary visual cortex (V1). The primary motor cortex (M1) is located in the dorsal portion of the frontal lobe. The primary somatosensory cortex (S1) is located in the postcentral part of the human brain. The primary auditory cortex (A1) is located bilaterally at the upper side of the temporal lobes on the human brain. The primary visual cortex (V1) is located above the brain stem.

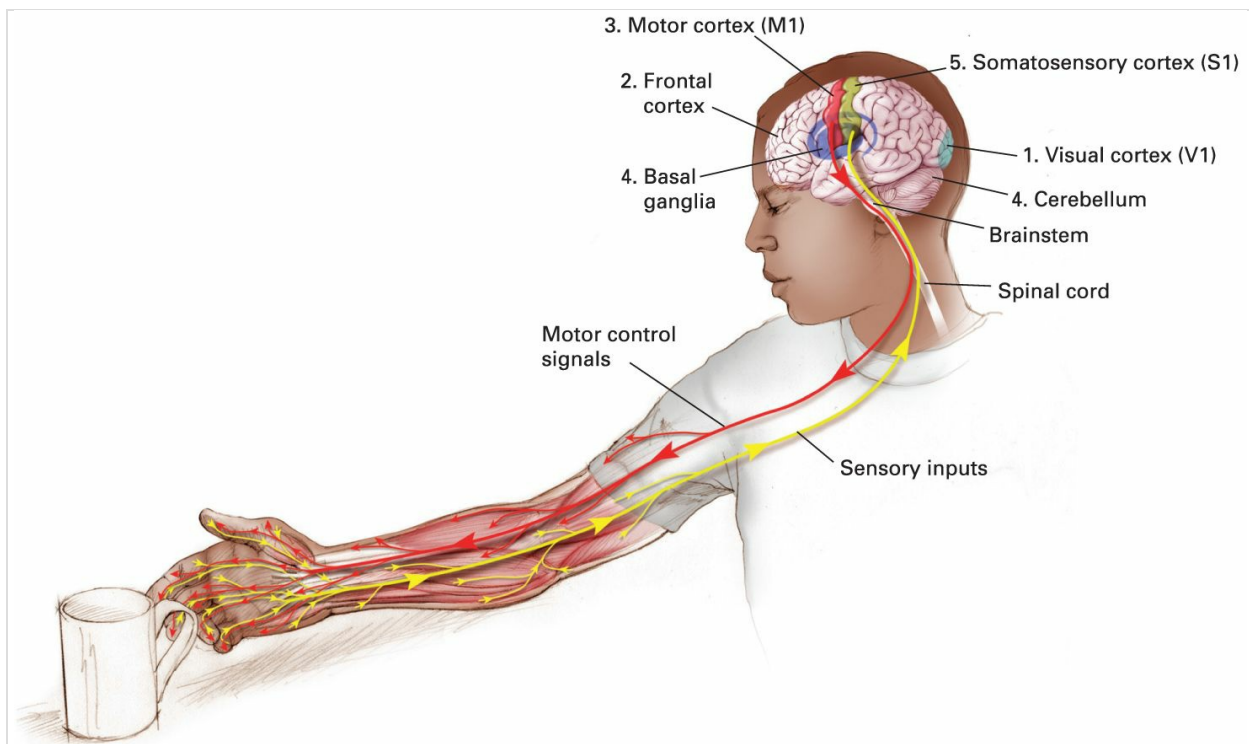
Outgoing Responses: Motor Control

Just as various brain regions are specialized for processing sensory inputs, other brain regions are specialized for processing the outputs that control movements. Chief of these is the primary

motor cortex (M1), which generates coordinated movements. M1 is located in the frontal lobe, adjacent to S1 in the parietal lobe ([Figure 2.8](#)), and it sends output to the brainstem, which in turn sends instructions down the spinal cord to activate motor fibers that control the muscles.

M1 gets much of its input from the frontal lobes, which are responsible for making high-level plans based on the present situation, past experience, and future goals. (Should you pick up that hot coffee cup? Should you try to catch that ball with one hand or two?) Other important inputs come from the basal ganglia and cerebellum, which help to translate the high-level plans into specific sets of movements. All these inputs help determine the outputs that M1 sends to the brainstem. Other motor areas—including the cerebellum, basal ganglia, frontal cortex, and the brainstem itself—also produce their own outputs, all of which converge on the spinal cord and travel from there to the muscles. Complex motor movements—such as picking up a hot coffee cup without spilling the liquid or burning your hand, or picking up an egg without crushing it, or dancing without stepping on your partner’s toes—require exquisitely choreographed interactions between all of these brain structures and the muscles they control.

Let’s consider one of these examples in greater detail: you see a cup of coffee and pick it up ([Figure 2.9](#)). The process begins with visual input from your eyes traveling to your visual cortex (V1), which helps you find and identify the cup. Regions in your frontal lobes coordinate the necessary plans for grasping the cup, which your motor cortex (M1) then directs by means of outputs through the brainstem, down sets of fibers in the spinal cord, and out to the muscles of the arm and fingers. As you reach for the cup, your basal ganglia and cerebellum continuously track the movement, making tiny adjustments as necessary. These brain regions enable you to exert just the right amount of pressure on the cup: enough to lift it against gravity but not so much that you yank it off the table and spill the contents. As you pick up the cup, sensory information from touch, heat, and pressure receptors in your fingers travels back up your arms, through sensory fibers in the spinal cord, and to the somatosensory cortex (S1), providing evidence that the cup is firmly in your hand. If the handle of the cup is hotter than expected, it could produce a reflexive withdrawal of the hand. This response is the kind of spinal reflex studied by Charles Sherrington; the short path from the hand to the spinal cord and back is sometimes called a *reflex arc* (a concept introduced by Descartes and described in [Chapter 1](#); compare [Figure 1.3](#) with [Figure 2.9](#)).



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FIGURE 2.9 How to pick up a cup of coffee (1) Visual input from V1 helps you locate the coffee cup and its handle. (2) The frontal cortex helps you plan the movement. (3) Outputs from the motor cortex (M1) travel through the brainstem and down sets of fibers in the spinal cord to the muscles in the arm, causing you to reach out your hand. (4) The basal ganglia and cerebellum continuously monitor whether your hand is on track, making tiny adjustments to ensure that your hand reaches the correct target. (5) Sensory signals travel back up the arm and spinal cord, through a second set of fibers, to the somatosensory cortex (S1), confirming that the cup has been grasped.

Description

The parts are numbered from 1 through 5.

The stimulus starts with the Visual cortex (1, V1). It then reaches the Frontal cortex (2), in the front brain, and continues to Motor Cortex (3, M1). Thereon, it is passed to the Basal ganglia (4) and moves to Somatosensory cortex (5, S1). The stimulus is then passed through the spinal cord. The motor control signals are transmitted from the brain through the spinal cord, to the hands of the man, and the sensory inputs are transmitted from the nerve ends in the fingers to the brain through nerves.

All that input and output just to pick up a cup—before you have even taken your first sip! Infants of many vertebrate species, including humans, are born fairly clumsy and spend a large part of their infancy and childhood learning how to walk or fly or swim gracefully, reach accurately, move throat and tongue muscles to produce coherent sounds, and so on. This relatively long

period spent learning coordinated motor control reflects both the complexity of the operations and the many brain structures that have to interact with one another and with the outside world to perform those operations.

Observing Brains in Action

Because learning is a process that can lead to changes in behavior, and brains control behavior through changes in neural activity, it is clear that learning must be associated with new patterns of activity in the brain. However, knowing that your brain is doing something different after years of practicing a skill or after experiencing a traumatic event is a far cry from knowing *what* it is doing differently or why. Even if structural imaging techniques reveal that experience has led to physical changes in parts of neurons or to increases in the volume of a brain region, understanding how these changes contribute to performance is not straightforward. To gain a clearer understanding of how experiences change brain function, neuroscientists attempt to monitor specific changes in activity that occur within the central nervous system before, during, and after such experiences.

Functional Neuroimaging

As noted earlier, structural neuroimaging methods (such as MRI) allow researchers to look at the *structure* of a living human brain, whereas [functional neuroimaging](#) allows them to look at the *activity*, or function, of a living brain. For example, when a brain structure becomes active, it requires more oxygen. Within 4 to 6 seconds, blood flow (with its cargo of oxygen) increases to that region. On the other hand, when a brain structure becomes less active, it requires less oxygen, and blood flow decreases. By tracking local changes in blood flow (or oxygen usage), researchers can infer which brain regions are more or less active.

functional neuroimaging

Techniques (such as fMRI) for observing the activity or function of a living brain.

Rather than focus on where blood use is heavier in the brain, functional neuroimaging studies typically examine how blood distribution or usage in a particular brain region *changes* depending on what the person is doing or thinking. To see such changes in blood distribution, researchers may first scan the brain while the person is relaxed and not doing anything. Of course, even though the person is not deliberately performing any task, the brain is still active. Next, the researchers scan the brain while the person performs a given task, such as looking at pictures or

reading a story. (The pictures or words are projected onto the inside ceiling of the scanner so that the person can see them while lying on his or her back.) During the task, some areas of the brain should become more active than before. Other brain areas might decrease in activity. The images are often color coded, with white, red, or yellow indicating areas where blood flow *increased* most during the task relative to the baseline. Colors such as blue and green may indicate where blood flow *decreased* most during the task.

Researchers typically do not rely on measurements from a single person to decide which brain regions are most likely to show changes in activity levels during performance of a particular task. Instead, they usually collect data from multiple individuals and then analyze patterns from the whole group. This approach emphasizes differences in brain activity that are prevalent across many participants, but it does not necessarily reveal all the changes in activity that occur within any particular individual. Through these methods, scientists can discover differences in brain activity that are associated with different kinds of memory tasks (for instance, recognizing faces versus recalling what happened at a recent party) and differences associated with successful recall versus memory failures.

The most commonly used functional neuroimaging technology is **functional magnetic resonance imaging (fMRI)**. It uses the same MRI technologies employed for structural imaging described in [Section 2.1](#), but with fMRI the focus is on differences in oxygen levels in the blood. Oxygenated blood produces slightly different signals than deoxygenated blood, so there are fluctuations in the signal received from areas of the brain that undergo changes in activity levels during task performance.

functional magnetic resonance imaging (fMRI)

A method of functional neuroimaging based on comparing an MRI of the brain during performance of a task with an MRI of the brain at rest.

fMRI is a powerful tool for observing the brain in action, but it only indirectly measures neural activity. Also, because functional neuroimaging studies typically focus on task-related changes in activity, they tend to emphasize associations between specific brain regions and particular functions (in the spirit of Gall) rather than look at the full range of brain activity that contributes to learning and memory processes. Finally, current functional neuroimaging techniques are relatively slow: fMRI allows images to be taken every few seconds, but changes in brain activity occur much more rapidly than that. Tracking learning- or memory-related changes in neural

activity in real time requires other technologies.

Electroencephalography

Electroencephalography (EEG) is a method of measuring electrical activity in the brain. (The Greek word *enkephalos* means “brain,” so “electro-encephalo-graphy” means drawing or graphing the electrical activity of the brain.) Electrodes placed on a person’s scalp measure the combined tiny electrical charges produced by millions of neurons, especially those near the location on the skull where the electrodes are placed. The resulting picture is called an *electroencephalogram* (also abbreviated as EEG).



Left: Aaron McCoy/Photolibrary/Getty Images; right: www.emotiv.com

To record neural activity from humans as they perform tasks, researchers attach multiple electrodes to a person’s scalp. Traditionally, signals from electrodes are collected from wires attached to the electrodes (as shown here for the infant), but recent wireless technologies make it possible to record neural activity in any setting. How might EEG be used to measure functional brain changes that have occurred in the adult model as a result of her modeling experiences?

Description

The photo on the left shows multiple electrodes and wires attached to a baby's head. The photo on the right shows electrodes and wires attached to a person’s scalp.

electroencephalography (EEG)

A method for measuring electrical activity in the brain by means of electrodes placed on the scalp; the resulting image is an *electroencephalogram* (also *EEG*).

Just as blood is always flowing through the brain, so is electrical activity, driven by the firing

patterns of neurons. The exact pattern of activity changes depending on what the brain is doing. For example, when a tone sounds, sensory receptors in the ear become active, and signals travel to the primary auditory cortex (A1), affecting electrical activity there. But detecting this particular electrical change in an EEG is difficult because lots of other neurons in other brain areas that are not involved in hearing may also be active—those responding to whatever visual stimuli happen to be in front of you, for instance, or those activated as you wiggle your fingers and think about what you want to have for lunch.

To detect an electrical change associated with hearing a sound, such as a tone, researchers typically present the same sound hundreds of times and then average the EEGs produced during those repetitions. Activity in other brain areas will come and go, but only the neurons responding to the specific sensory stimulus will be consistently activated each time the sound is repeated—and so only their activity patterns will survive the averaging process.

EEGs averaged across many repetitions of the same event are called **event-related potentials (ERPs)**. Just as functional neuroimaging shows how the brain changes while performing a task, ERPs can be used to show different brain states at different stages of learning or memory processing, such as how a person's brain responds to different sounds as the person gradually learns to make subtle distinctions between them (discussed in [Chapters 3](#) and [6](#)).

event-related potential (ERP)

Electroencephalograms (EEGs) from a single individual averaged over multiple repetitions of an event (such as a repeated stimulus presentation).

Compared with fMRI, EEG recording is a simple and inexpensive way to monitor changes in brain activity during learning and memory tasks. In addition, EEG can detect rapid changes in the brain with more temporal precision than fMRI. Yet what EEG gains in temporal precision, it often sacrifices in spatial precision. Whereas fMRI can localize activation to within a few millimeters, EEG signals show activity over a wide swath of the brain. To take advantage of the strengths of both technologies, some memory researchers are combining functional neuroimaging and EEG methods to generate images that show precisely when and where neural activity occurs during memory storage and recall by humans.

Recording from Neurons

Memory functions are affected not only by *which* neurons fire but also by *how often* they fire. Neuroimaging and EEG studies can reveal the contributions of large areas of the brain to learning and memory, but they do not reveal much about which individual neurons are firing or how often. To gather this information, researchers have to record neural activity directly.

Neurophysiology is the study of the activity and function of neurons.

neurophysiology

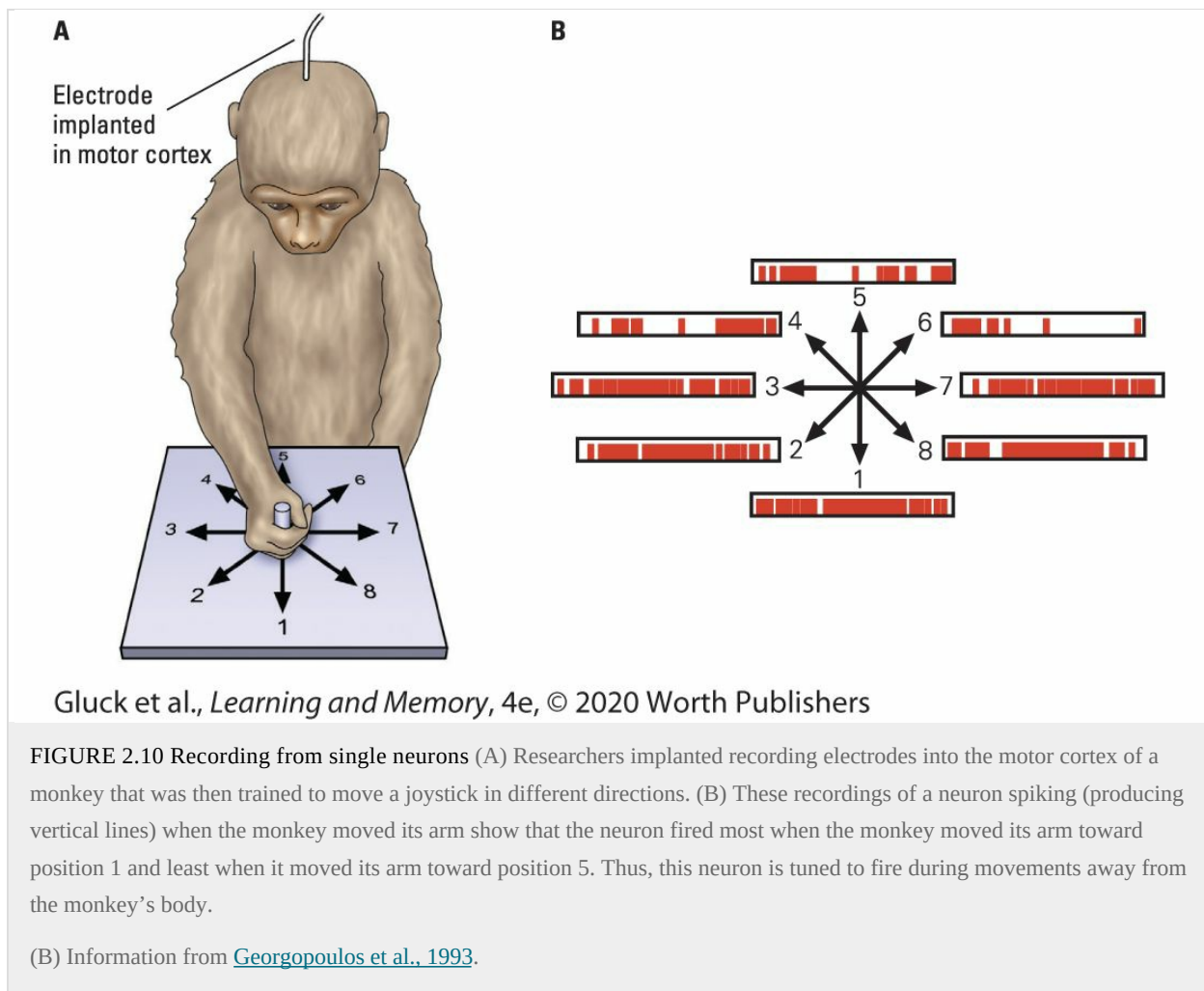
The study of the activity and function of neurons.

One technique scientists use to measure the firing patterns of individual neurons is **single-cell recording** (where the single cell in this case is a neuron). The microelectrodes that are employed in this method function somewhat like EEG electrodes, but they are much tinier and can penetrate brain tissue without too much damage. A microelectrode can be inserted in brain tissue until its tip is very close to, or sometimes even inside, a target neuron. In some cases, researchers anesthetize an animal and surgically implant one or more microelectrodes in the brain areas they wish to study. Then, when the animal wakes, the researchers can record from the neuron(s) as the animal goes about its daily business. Most animals do not seem to be much bothered by, or even aware of, the wires connected to their heads. Such experiments allow researchers to determine what role a given neuron or network of neurons might play in the animal's behavior. Alternatively, if the researcher is interested in looking more closely at how individual neurons interact, it is possible to remove pieces (or "slices") of a brain, keep the neurons alive in a bath of nutrients, and record their activity.

single-cell recording

Use of an implanted electrode to detect electrical activity (spiking) in a single cell (such as a neuron).

Single-cell recordings have provided some of the most dramatic evidence to date of how neural firing relates to behavior. For example, Apostolos Georgopoulos and colleagues recorded neural firing patterns from the motor cortex of a monkey while the monkey moved a joystick in different directions (**Figure 2.10A**; [Georgopoulos, Taira, & Lukashin, 1993](#)). Some neurons fired most strongly when the monkey pushed the lever in a particular direction. [Figure 2.10B](#) shows recordings from one such neuron as the monkey moved the lever toward different compass points.



Description

Section A shows a drawing of a monkey with an electrode implanted in the motor cortex. The monkey is working a joystick that has positions 1 through 8 marked radially around it.

Section B shows 8 positions of the joystick and the recorded response rate associated with each position. Spikes represent the response rate. Position 1, at the bottom, has the most spikes. The spikes grow fewer as the joystick moves away from Position 1.

Each vertical line in the recording shown in [Figure 2.10B](#) represents one action potential, sometimes referred to as a *spike*. When the monkey moved its arm toward the point labeled 6 in [Figure 2.10A](#), the neuron initially produced several spikes, and then it fell silent. When the monkey moved its arm to a slightly different position, point 7, the neuron produced a more sustained burst of activity, and it continued to spike for the duration of the movement. But when the monkey moved its arm directly away from its body, toward point 1, the neuron really went

into action, spiking as fast and frequently as it could. By contrast, when the monkey moved its arm in the opposite direction, toward its body (point 5), the neuron was much less active. Thus, this neuron's firing patterns are correlated with arm movements, and neuroscientists would say it is specialized, or “tuned,” to fire maximally during movements in a particular direction: away from the body.

Georgopoulos and colleagues found that other neurons in the motor cortex are tuned to fire during arm movements in other directions. Given what we know about the motor cortex from functional imaging studies, it is reasonable to assume that these neurons may be playing a direct role in issuing the commands that cause the monkey's arm to move. Because monkeys generally must be trained to move a joystick in different directions in laboratory experiments, such recordings can potentially reveal how the firing patterns of neurons change as monkeys learn to perform such tasks. In fact, such research has led to new technologies that enable both monkeys and humans to learn to control the movements of robotic arms simply by thinking about where they want the arm to move (discussed in [Chapter 8](#)).

Single-cell recordings can also be collected from humans when doctors need to clinically monitor patients with epilepsy before performing corrective neurosurgery. This practice makes it possible to observe neural activity as a person remembers specific episodes ([Suthana & Fried, 2012](#)).

How Remembering Changes Brain Activity

At the start of this chapter, we described the case of Orlando Serrell, who gained remarkable memory abilities after sustaining a blow to the head in a baseball game. Orlando is not the only person to possess phenomenal memory capacities. By studying people with an exceptional ability to remember specific events, researchers can gain unique insights into the systems that make memory possible.

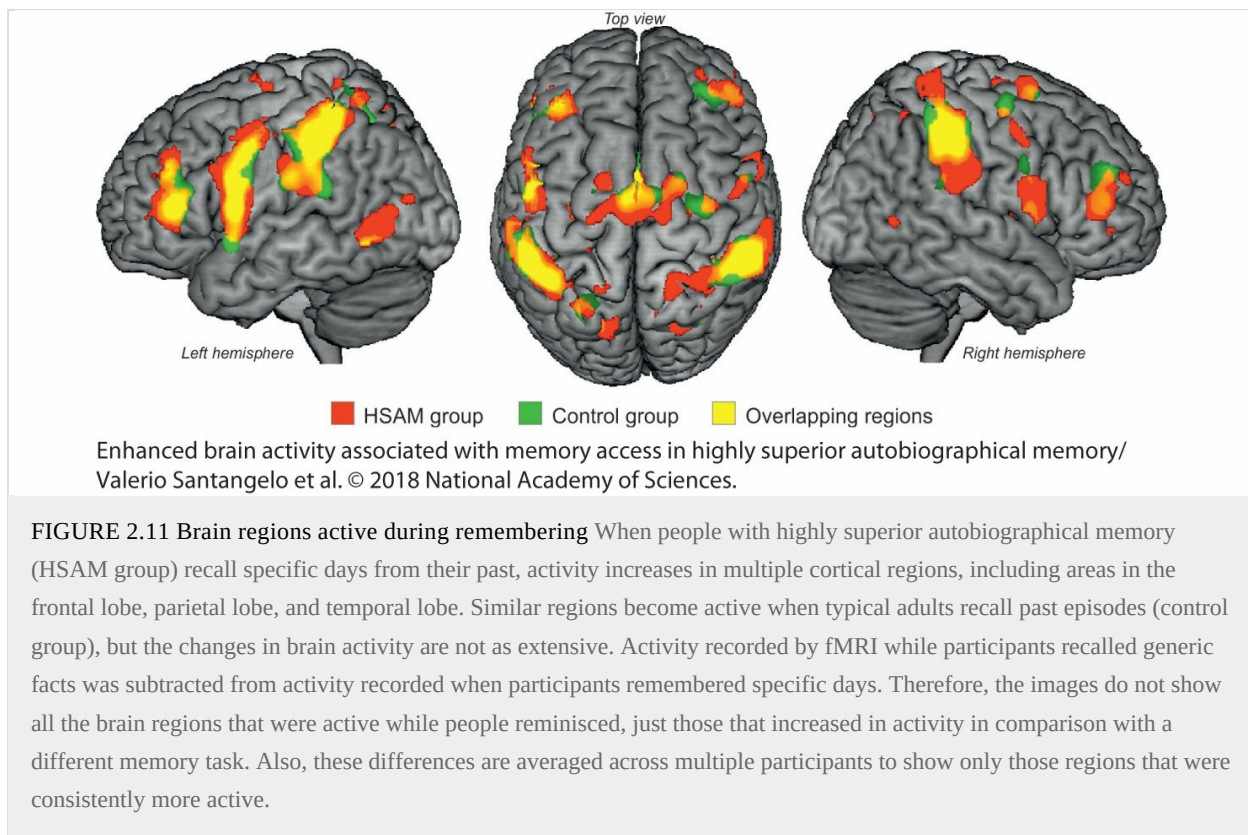
Some people who excel at remembering compete in annual competitions to see who can memorize the most information quickly and accurately—things like sequences of numbers or playing cards, names and faces, and so on. These memory champions have trained their brains to absorb details and retain them at least long enough to recite or write them down during the recall portion of the contest. Most such memory experts have learned to use specific strategies or

mnemonics to do this (discussed in [Chapter 7](#)). But memory savants, like Orlando, seem to perform amazing feats of memory involuntarily, without needing to practice, and their memories are typically for very different kinds of information than the items competitors excel at memorizing.

One group of memory savants that has received attention in the past decade is made up of people with *highly superior autobiographical memory (HSAM)*, or *hyperthymesia* ([LePort et al., 2012](#)). These individuals appear to be able to access memories of events that happened on almost any day of their lives and can recall seemingly trivial details such as the meals they ate each day. Think about what you did yesterday: whom you met, what you ate, where you went, and what you wore. Now imagine being able to remember any day from the past 10 years with the same level of detail. This is what people with HSAM seem to be doing just as easily as you can envision yesterday. For other kinds of memory, though, people with HSAM perform like everyone else. They do not have “photographic memories.”

So, what is different about the brains of people with HSAM? Are they experiencing events in ways that make those events more memorable than they would be for the typical person? Or perhaps their experiences are normal but they have an exceptional ability to access and relive memories of those experiences. It is hard to tell based only on their abnormal accuracy or on their descriptions of what remembering is like for them. Recent functional neuroimaging studies provide a few clues, however, about what is going on in their brains. To see what is happening in people with HSAM when they remember past events, researchers gave them prompts like “the first time you drove a car” and then monitored their brain activity using fMRI while the episode was being remembered ([Santangelo et al., 2018](#)). They then compared the activity recorded during the remembering of autobiographical events with brain activity that occurred in response to prompts like “examples of vegetables.”

The researchers discovered that when remembering past events, the savants showed increased activity in several different brain regions, especially the *prefrontal cortex* (a subregion of the frontal lobe discussed in [Chapters 7](#) and [9](#)), the hippocampus, and a region of cortex at the junction of the frontal and parietal lobes ([Figure 2.11](#)). Interestingly, the specific patterns of activation varied considerably across individuals with HSAM, contrary to what you (or Gall) might expect if specific regions of cortex were specialized for remembering personal episodes.



Description

The first illustration shows the left hemisphere. Overlapping regions cover the major parts of the left hemisphere.

The second illustration shows the top view. The overlapping regions and the other highlighted regions are spread throughout.

The third illustration shows the right hemisphere. The overlapping regions are at the top center part of the brain. The other highlighted regions are in the anterior part of the brain.

Comparing brain activity during recall of personal episodes to brain activity during recall of facts shows that something different is going on when people with HSAM remember things that happened to them. But is this true only for people with HSAM? Would you see the same kinds of differences if you scanned your own brain activity? To learn whether that might be the case, researchers conducted the same fMRI study using a control group consisting of people without super memory. This comparison revealed that people with HSAM were recruiting more cortical regions than the control participants were using when remembering experienced events ([Figure 2.11](#)). However, this difference showed up only when participants were initially accessing the personal memories. When participants went on to think about the details of the memory, brain

activity was comparable for both groups. This could mean that the way people with HSAM access past personal memories is somehow unique.

One complication of simply asking people to “think back” to events that may be long past is that it can be difficult to know how accurate a person’s memory for a particular day really is. A memory may feel like a true record of what really happened, but feelings of certainty are no guarantee of accuracy. ([Chapter 7](#) has more to say about *false memories* that feel more real than they are.) New personal recording technologies like GoPro enable people to document episodes in their lives, and researchers are beginning to incorporate such devices into fMRI studies, to help trigger and control the personal memories of research participants ([Chow & Rissman, 2017](#)). Segments of scenes recorded from the viewpoints of study participants or of other individuals can be used to prompt recall of specific episodes and to test participants on their ability to tell whether an event was one they personally experienced. Such tasks activate networks of brain regions that are similar to those seen in people attempting to recall episodes such as their first kiss, providing further support for the idea that many different brain regions work together to make remembering the past possible.

Interim Summary

- Reflexes are natural, automatic responses to stimuli. Sherrington and other early neuroscientists believed that all complex learning involves combining simple spinal reflexes.
- In the brain, sensory signals (produced by stimuli) are initially processed in cortical regions specialized for processing such signals, and they ultimately lead to activity in other cortical regions, such as the motor cortex, that are specialized for coordinating movements (responses).
- Functional neuroimaging methods (such as fMRI) allow researchers to track brain activity during the performance of memory tasks by measuring increases and decreases in activity in different brain regions.
- Electroencephalographic recordings make it possible to track the activity of large populations of neurons over time and to monitor how such activity changes as learning progresses.
- Single-cell recordings allow researchers to directly monitor and record the electrical activity (or “firing”) of single neurons and to study changes in their firing patterns that occur during learning or the recall of memories.

- Brain-imaging studies of memory savants and others show that the act of remembering experienced events engages a broad network of brain regions.

2.3 Finding and Manipulating Memories

We have discussed two ways in which experience can change the brain: structurally, by changing properties of neurons and their connections; and functionally, by changing patterns of activity in neural circuits. These two kinds of change are closely related because changes in neural activity can lead to physical changes in brain tissue (as proposed by Ramón y Cajal and James), and physical changes partly determine future patterns of neural activity.

We have also touched on the idea that different brain regions may be specialized for different kinds of learning or memory functions (an idea that Gall got mostly right!). Baby chicks show localized structural changes in their brains when they imprint on their mothers, and undergraduates show localized changes in brain activation when they recall episodes from their personal lives. Such findings might give you the impression that neuroscientists have solved the riddle of how brains store memories. Some do make this claim, but unfortunately, they do not all agree on what the answer is. Debate rages on about how memories are stored by the brain and especially about *where* they are stored.

Looking for Memories

Most neuroscientists are on board with the idea that memories are stored in certain brain regions, and most assume that within those brain regions, memories are stored by physical properties of neurons. There are differing opinions, however, about where specific kinds of memories might be stored, how long they might be stored in a particular location, whether they might change location over time, and whether different people might store them in different locations. Similarly, there are several different proposals about what parts of neurons, or sets of neurons, are the parts that store the memories. Two themes that have dominated research on the brain mechanisms that support learning and memory (in other words, on the neural substrates of learning and memory) are that many memories are stored in the cerebral cortex and, more specifically, that they are stored in connections between cortical neurons.

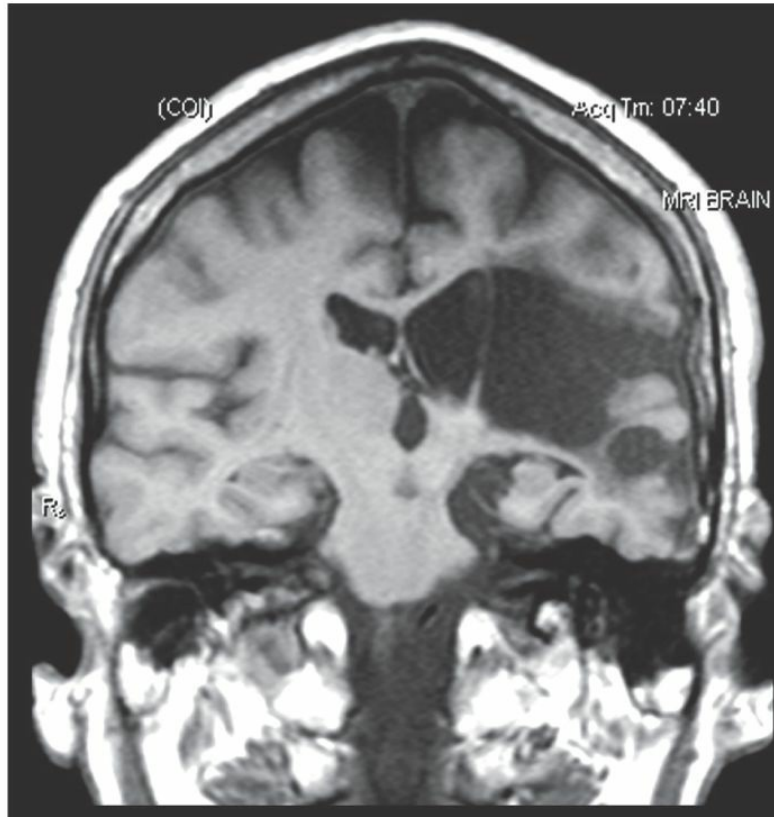
Searching for Memories in the Cerebral Cortex

Imagine that a Martian scientist comes to Earth and encounters an automobile, a method of transportation unknown on Mars, powered by an energy source also unknown to Martians. If the Martian speaks no Earth languages and can't simply ask a mechanic for an explanation, how

might she learn how the car works? One way would be to look under the hood and examine the many components there. But studying the car's "structure" would only get her so far; to learn about the car's function, she would have to take it for a test drive and see how it behaves when it is operating properly. However, simply seeing cars in action cannot reveal what makes them go.

One approach the Martian might use to better understand cars would be to investigate what the different parts do. For instance, she could try disconnecting or removing parts one at a time, noting the consequences in each case. If she removes the axle, she will find that the motor works but cannot transfer energy to make the wheels turn. If she removes the radiator, she will find that the car runs but then quickly overheats. In the end, by discovering the function of each of the car parts, the Martian could probably develop a pretty good idea of how the car works.

Neuroscientists trying to understand how nervous systems make it possible for organisms to learn and remember face a challenge similar to the Martian's. No surprise, then, that one of the earliest approaches researchers attempted was akin to the Martian's: they examined people whose brains were missing pieces to see how such losses could affect performance. Although no scientist would disassemble the brain of a living human the way the Martian might disassemble a car, humans regularly suffer damage to one or more brain areas—through accident, injury, or disease—making it possible to explore the effects that damaged brain regions can have on learning and memory abilities.



Living Art Enterprises/Science Source

Brain injuries can lead to the loss of large portions of brain tissue. In this MRI image, missing cerebral cortex appears as a dark region on the right side of the image. Is this lesion more likely to be in the temporal lobe or the occipital lobe?

Neuropsychology is the branch of psychology that deals with the relationship between brain function and behavior, usually by examining the functioning of patients who have specific types of brain damage. These individuals volunteer their time and effort in experiments that test their learning and memory abilities, as well as other kinds of cognitive function—language, attention, intelligence, and so on. The test results can potentially be used to guide a patient’s rehabilitation, but they also serve a research purpose. By recognizing patterns in the impaired and spared abilities of a group of patients who have experienced damage to a similar region of the brain, researchers hope to build a better picture of that brain region’s normal function—just like the Martian trying to understand what a radiator does by watching what happens to a car that does not have one.

neuropsychology

The branch of psychology that deals with the relationship between brain function and behavior.

Animal researchers have conducted parallel studies by removing or deactivating specific brain regions to create animal “models” of humans with brain damage. Because human brain damage is almost always caused by accident, injury, or illness, every patient’s damage—and disability—is slightly different. By contrast, in animal models, researchers can remove or disable specific brain regions with great precision, making it much easier to compare results across individuals. Instances in which the experimental results from human patients and animal models converge give the clearest picture of how the brain works normally and how it functions after damage.

Some of the most famous experimental brain lesion studies of learning and memory were conducted by Karl Lashley (1890–1958), an American psychologist who was looking for the location of the **engram**—the supposed physical change in the brain that forms the basis of a memory (also referred to as a *memory trace*). Lashley would train a group of rats to navigate a maze, and then he would systematically remove a different small area (covering, say, 10%) of the cortex in each rat. He reasoned that once he had found the lesion that erased the animal’s memories of how to run through the maze, he would have located the site of the engram ([Lashley, 1929](#)).

engram

A physical change in the brain that forms the basis of a memory.

Alas, the results were not quite so straightforward. No matter what small part of the cortex Lashley lesioned, the rats kept performing the task. Bigger lesions would cause increasingly large disruptions in performance, but no one cortical area seemed to be more important than any other. Hence, Lashley could not find the engrams for memories formed during maze learning. Finally, in mock despair, he confessed that he might be forced to conclude that learning “simply is not possible” ([Lashley, 1929](#)).

Eventually, Lashley settled on a different explanation. He endorsed the **theory of equipotentiality**, which states that memories are not stored in one area of the brain; rather, the brain operates as a whole to store memories. Although Lashley is often credited with formulating this theory, it was actually first proposed in the 1800s as an alternative to phrenology ([Flourens, 1824](#)). In the theory of equipotentiality, memories are spread over many cortical areas; damage to one or two of these areas will not completely destroy the memory, and over time the surviving cortical areas may be able to compensate for what has been lost.

theory of equipotentiality

The theory that memories are stored globally by the brain as a whole rather than in one particular brain area.

Lashley's work and his endorsement of the theory of equipotentiality were milestones in the neuroscience of memory because researchers could no longer take for granted the compartmentalized cortical regions that Gall had popularized. But Lashley was only partly correct. Gall was on the right track when he proposed that different brain areas have different specialties; the specialization just wasn't as extreme as he had thought. Lashley, too, was on the right track when he proposed that engrams are not stored in specific areas of the cortex, but we now know that the cortex is not quite as flexible as he came to believe. The truth is somewhere in the middle.

Part of the reason Lashley's experiments did not work out the way he expected was because of his assumption that memories formed during maze learning were stored only in the cerebral cortex. If Lashley had instead made his lesions beneath the cortex, he might have discovered that other brain regions (such as the hippocampus) more strongly affect spatial learning and memory. (The role of the hippocampus in spatial learning is discussed in [Chapters 3](#) and [7](#).)

Useful as brain lesion experiments are, they are limited in terms of what they can reveal. Suppose a researcher lesions part of a rat's cortex and then finds, as Lashley did, that the rat can still learn how to get around in a maze. Would that prove that the lesioned cortical area is not involved in spatial memory? Not necessarily; the rat may now be learning the maze in a different way. This would be analogous to your being able to find your way around a house with the lights out, even though you use visual input when it is available. Data from lesion studies are strongest when supplemented by data from other techniques showing that a brain region normally participates in a given behavior.

Showing that a particular brain region needs to be intact for learning and memory processes to work normally also does not mean that memories are stored in that region. Your laptop's memory will not work without a power source, but that does not mean it is storing memories in its battery or power cable. To establish that a memory has been stored in a particular part of your laptop, you would need to show that specific kinds of changes in that part always occur when such a memory is stored; that the changes remain stable as long as the memory is stable; that the changes are specific to the content of the memory that was stored; and that when the memory is

retrieved, retrieval processes access the changes made in that part. Researchers use similar tests to try to identify engrams in brains. Usually, these tests focus specifically on the role of physical changes that occur along dendrites.

TEST YOUR KNOWLEDGE

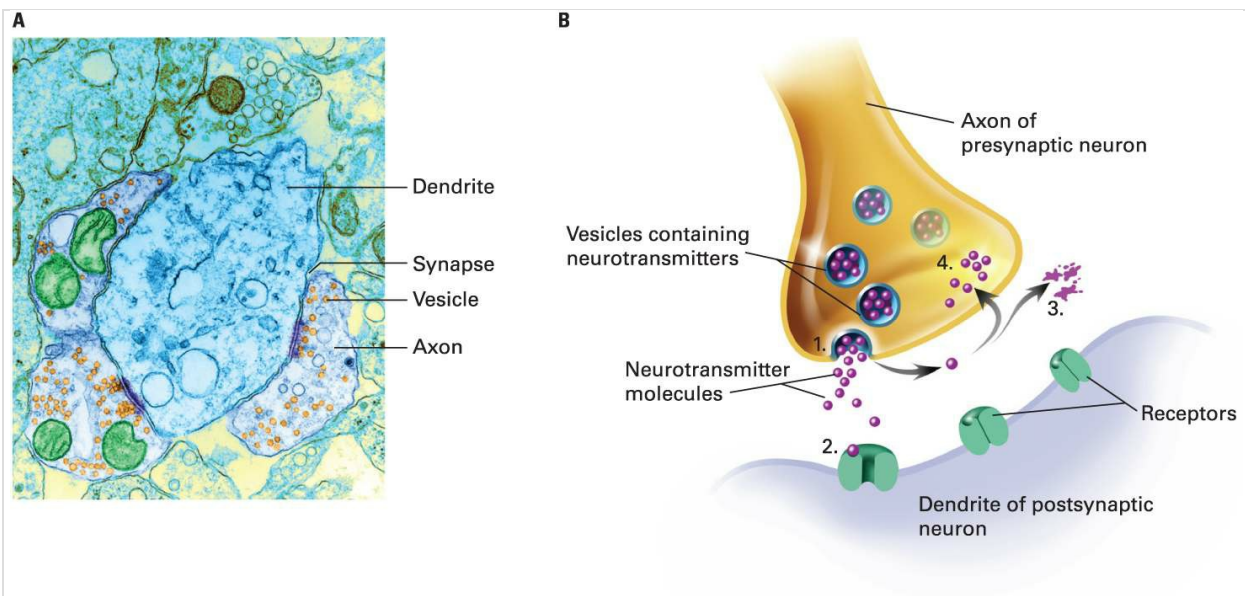
Equipotentiality Versus Phrenology

What are the main differences between the explanations of brain function proposed by Franz Joseph Gall and those ultimately proposed by Karl Lashley? What evidence did each use to support his viewpoint? ([Answers appear at the end of the chapter.](#))

The Synapse: Where Neurons Connect

So far, we have been describing the transmission of signals into and out of the brain as if these signals flow from one place to another in the nervous system like water through a pipe (reminiscent of the way Descartes described the mechanisms of behavior). What really happens is that neurons throughout the nervous system are continually communicating with one another in vast networks that are similar in some ways to social networking systems such as Twitter or Facebook. This communication between neurons makes learning and memory possible.

Generally, neurons that communicate with each other are not actually physically connected. Rather, communicating neurons are separated by a narrow gap of about 20 nanometers (1 nanometer is one-billionth of a meter), called a **[synapse](#)**, across which the neurons pass chemicals (**[Figure 2.12A](#)**). Most synapses are formed between the axon of the **[presynaptic](#)** (or sending) neuron and a dendrite of the **[postsynaptic](#)** (or receiving) neuron, but synapses can also be formed between an axon and a cell body, between an axon and another axon, and even between dendrites.



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

A: OMIKRON/Science Source

FIGURE 2.12 Transmission across a synapse (A) This photo (taken through an electron microscope) shows the tiny gaps, or synapses, between neurons. Vesicles filled with neurotransmitters, ready for release into the synapse, are visible as circular packets inside the presynaptic neuron. (B) A signal is transmitted between neurons when (1) the presynaptic neuron releases neurotransmitter into the synapse and (2) the neurotransmitter molecules dock at receptors on the surface of the postsynaptic neuron. This may activate the receiving neuron. Leftover neurotransmitter in the synapse is either (3) broken down or (4) reabsorbed into the presynaptic neuron.

Description

Section A shows a micrograph with the following parts labeled: Dendrite, Synapse, Vesicle, and Axon.

Section B shows the release of neurotransmitter molecules from a presynaptic neuron. The regions labeled are as follows. Axon of presynaptic neuron lies at the tip of the neuron. The rounded structures within the axon of the presynaptic neuron are labeled vesicles containing neurotransmitters. The steps are numbered from 1 through 4. The vesicles along the bottom periphery of the axon are open, and the neurotransmitter molecules are released from it (1). These molecules move toward the receptors in the area labeled dendrite of the postsynaptic neuron. The receptors accept the neurotransmitter molecules (2). The unaccepted neurotransmitter is broken down (3) or reabsorbed (4).

synapse

A narrow gap between two neurons, across which chemical messages can be transmitted.

presynaptic

On the sending side of a synapse.

postsynaptic

On the receiving side of a synapse.

Neurons contain **neurotransmitters**, chemical substances that can cross a synapse to affect the activity of a postsynaptic neuron. Neurotransmitters are kept conveniently on hand at the end of

the presynaptic axon, in packets known as *vesicles*. To transmit a signal, one or more vesicles of the presynaptic axon release neurotransmitters into the synapse ([Figure 2.12B](#)). Several different chemicals act as neurotransmitters. Major ones include *glutamate*, *gamma-aminobutyric acid* (GABA), *acetylcholine*, *dopamine*, *norepinephrine*, *epinephrine*, and *serotonin*. Once neurotransmitters have been released into the synapse, the next step is for the postsynaptic neuron to collect them. **Receptors** are molecules embedded in the surface of the postsynaptic neuron that are specialized to bind with and respond to particular kinds of neurotransmitters.

neurotransmitter

One of several classes of molecule released by neurons to carry chemical messages to other neurons.

receptor

A specialized molecule, located on the surface of a neuron, to which one or more particular neurotransmitters can bind; when a neurotransmitter activates a receptor, effects may be initiated in the neuron.

The effect of a particular neurotransmitter depends on what its corresponding postsynaptic receptors do when activated. Some receptors open a channel for the flow of electrically charged particles into or out of the cell, thus changing the charge characteristics in a small area of the neuron. Similar electrical changes may be occurring simultaneously in other locations on the neuron as other receptors on other dendrites become active. When a neuron fires, sending an electrical charge to the end of its axon, it causes the release of neurotransmitters there.

Some neurotransmitters—glutamate, for example—are *excitatory*, activating receptors that tend to increase the likelihood of the postsynaptic neuron firing. Other neurotransmitters—such as GABA—are *inhibitory*, activating receptors that tend to decrease the likelihood of the postsynaptic neuron firing. A neuron may produce and release only one kind of neurotransmitter or may release more than one kind. In either case, the neuron may be able to respond to signals from many different presynaptic neurons, each releasing a different kind of neurotransmitter.

After a neuron fires, there is a brief period, called a *refractory period*, during which it cannot fire again, no matter how much input it receives. When this refractory period has passed, the neuron is again open for business. If the neuron is still receiving a lot of input from its neighbors, it may fire again and again in rapid succession, interrupted only by the refractory period after each action potential. If the excitatory inputs are less frequent or less strong, or if there is a lot of inhibitory input, some time may pass before the neuron fires again.

In the meantime, neurotransmitter molecules have to be cleared out of the synapse so that the synapse can receive future signals. In some cases, this consists of breaking down the neurotransmitter molecules into their constituent parts through a process called *inactivation*. In other cases, they are brought back into the presynaptic neuron and recycled for future use in a process called *reuptake*. When cleanup is complete, the synapse and receptors are ready to receive new transmissions.

Several areas in the brainstem contain neurons that send axons widely throughout the brain; when these neurons fire, they release neurotransmitters called **neuromodulators** that can affect activity in entire brain regions rather than just at a single synapse. Neuromodulators alter, or modulate, how neurons transmit and receive signals, although they themselves are not part of the signal. For example, acetylcholine often functions as a neuromodulator, and one of its effects is to temporarily alter the number of receptors that must be active before a postsynaptic neuron can fire. If you think of synaptic transmission as a message, then acetylcholine levels help determine whether the message is heard as a whisper or a shout. Many human diseases that affect learning and memory seem to involve a global decline in neuromodulators. Examples include Alzheimer's disease, which is associated with a reduction in acetylcholine ([Francis, Palmer, Snape, & Wilcock, 1999](#)), and Parkinson's disease, which is characterized by a reduction in dopamine ([Evans & Lees, 2004](#)).

neuromodulator

A neurotransmitter that acts to modulate activity in a large number of neurons rather than in a single synapse.

All of these processes depend on the structure of neurons, axons, dendrites, and synapses, and all can potentially be changed by learning experiences. Nevertheless, learning and memory researchers have focused almost exclusively on understanding the role of **synaptic plasticity**, the ability of synapses to change as a result of experience.

synaptic plasticity

The ability of synapses to change as a result of experience.

TEST YOUR KNOWLEDGE

Synaptic Transmission

Several complex processes that occur at the synapse allow neurons to communicate. Which, if any, of the statements below do *not* describe one of these processes? ([Answers appear at the end of the chapter.](#))

1. Neurotransmitters are reabsorbed by the axon that released them.
2. Neurotransmitters are broken down while they remain in the synapse.
3. Neurotransmitters bind to the dendrite of the postsynaptic neuron.
4. Neurotransmitters are released from vesicles and enter the synapse.

Long-Term Potentiation

In the late 1960s, Terje Lømo was pursuing his doctoral degree in the lab of Per Andersen at the University of Oslo in Norway. Part of Lømo's research involved electrically stimulating the axons of presynaptic neurons that provided inputs to the hippocampus of a rabbit. He simultaneously recorded electrical activity produced by postsynaptic neurons within the hippocampus ([Figure 2.13A](#)). Normally, a certain amount of stimulation produced a certain level of response: a single weak stimulation would produce a low response in hippocampal neurons, and a strong burst of high-frequency stimulation (say, 100 stimulations in a second) would produce a more robust response. But to Lømo's surprise, the high-frequency stimulation also caused a lasting change in responding, so that hippocampal neurons would over-respond to subsequent weak stimulation ([Figure 2.13B](#)). This change could last for hours ([Bliss & Gardner-Medwin, 1973](#); [Bliss & Lømo, 1973](#); [Lømo, 1966](#)).

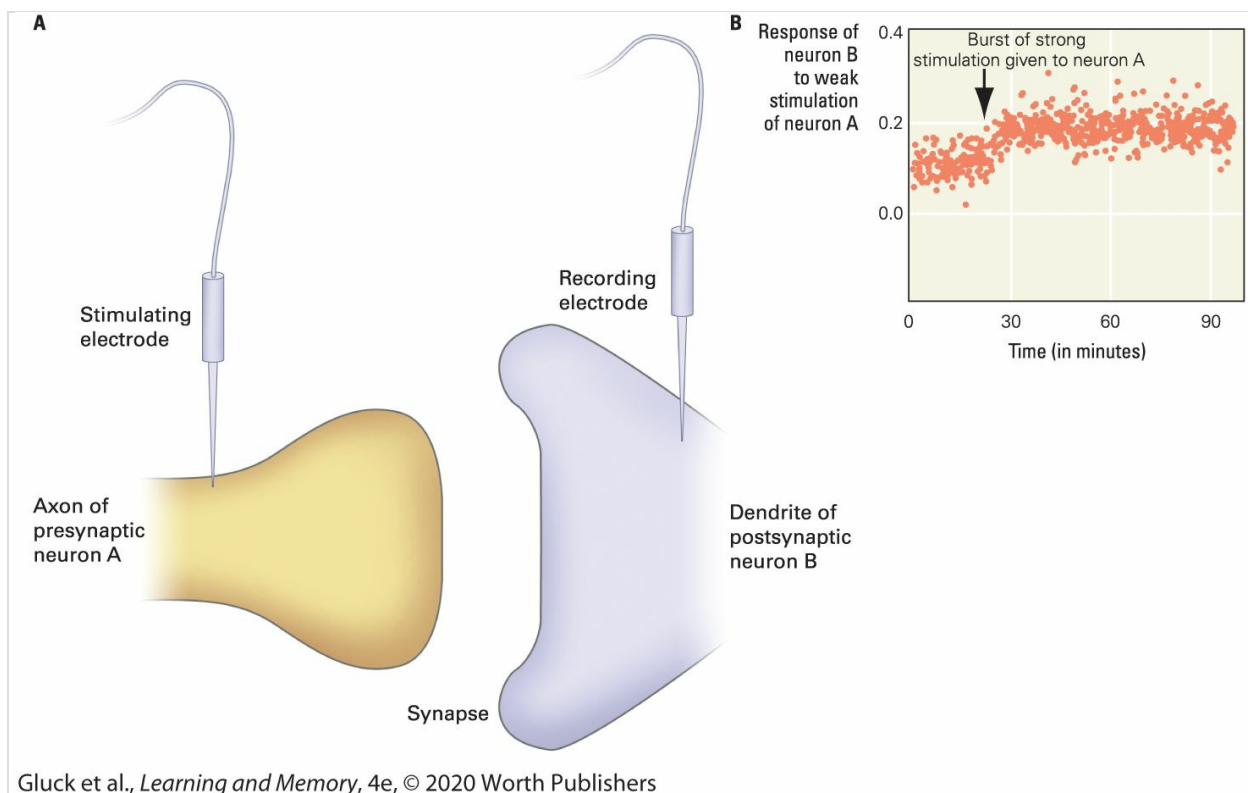


FIGURE 2.13 Long-term potentiation (LTP) (A) In the original LTP studies, researchers used an electrode to stimulate the axons of presynaptic neurons (here depicted as the axon of a single neuron A) and a second electrode to record the activity of postsynaptic neurons (here shown as the dendrite of a single neuron B). (B) Initially, weak stimulation caused a mild response in postsynaptic neurons. But after a burst of high-frequency stimulation, postsynaptic neurons persistently responded more strongly to the weaker stimulation.

Description

Section A shows the axon of presynaptic neuron A positioned closely to a dendrite of postsynaptic neuron B. The axon of neuron A is connected to a stimulating electrode and the dendrite of neuron B is connected to a recording electrode.

The graph in section B plots the response of neuron B to weak stimulation of neuron A on the vertical axis against time in minutes on the horizontal axis. The readings on the vertical axis range from 0 to 0.4 in increments of 0.2 and the readings on the horizontal axis range from 0 to 90 minutes in increments of 30. The plots corresponding to a response of 0.0 to 0.2 and a period of 0 to 90 minutes mark the burst of strong stimulation given to neuron A, after which the plots plateau.

Imagine that you have a brother who constantly torments you with his snide comments. Most of the time, you don't react. But one day he says something that is really over the top, and you respond with some strong language of your own. A few minutes later, before you have had a chance to calm down, he makes another little snide comment. Ordinarily, you might not have bothered to respond. But this time, you haven't yet cooled down from the earlier explosion, so you lose it again. Your prior anger has *potentiated* your response to a weak stimulus that normally would not have evoked such a strong reaction.

Potentiation of a neuron by a strong stimulus is similar (except that the “irritation” can last much longer), making the neuron more likely to respond to any subsequent stimulus. This effect, in which synaptic transmission becomes more effective as a result of recent activity, came to be called **long-term potentiation (LTP)**. The reports by Lømo and his coworkers demonstrated not only that electrical stimulation could be used to cause neurons to change their activity but also that these changes could last for hours or even days ([Bliss & Gardner-Medwin, 1973](#); [Bliss & Lømo, 1973](#)). Since that time, LTP has become one of the most extensively investigated phenomena in the neuroscience of memory. LTP is widely viewed as a mechanism of Hebbian learning that brains use to store memory traces in synaptic connections between neurons.

long-term potentiation (LTP)

A process in which synaptic transmission becomes more effective as a result of recent activity; with long-term depression, LTP is widely believed to represent a form of synaptic plasticity that could be the neural mechanism for learning.

LTP is also closely aligned with the kinds of *associative models* proposed by William James, as well as the *connectionist models* developed by David Rumelhart and James McClelland (described in [Chapter 1](#)). Both types of models describe links between processing units, or *nodes*, that change in strength depending on the specific memories that are being stored. If you think of these links as being analogous to cartoon versions of synaptic connections, then you can imagine that adjusting lots of little links might lead to unique constellations of linked nodes (*neural networks*). Psychologists often use connectionist models to simulate learning and memory processes; several examples appear in later chapters.

Despite intensive study of LTP in the decades since it was initially reported, many questions remain about exactly what changes occur to produce it, as well as how it relates to learning in more natural contexts. Perhaps postsynaptic neurons change to become more responsive to subsequent inputs. This would mean that when presynaptic neurons release neurotransmitters after strong stimulation, the postsynaptic neurons will have heightened sensitivity to that neurotransmitter, producing the enhanced response seen in [Figure 2.13B](#).

LTP may also involve changes to presynaptic neurons. This idea is controversial because it is not clear exactly how signals could travel backward across the synapse. But perhaps some kind of chemical—a *retrograde messenger*—could be released by postsynaptic neurons and diffuse back across the synapse, altering the amount of neurotransmitter the presynaptic neurons release in the future. These changes might occur within a few minutes and could last several hours. In addition, most researchers currently believe there are components of LTP that take place over several hours and can last a lifetime. This would involve changes such as strengthening of existing synapses or even building of new ones ([Chen, Rex, Casale, Gall, & Lynch, 2007](#)).

As excited as researchers were about the possibilities of LTP as a mechanism for learning and memory, they were aware of a significant remaining question. LTP represents a way to strengthen neural connections, but strengthening alone is not much use. If you think of the activity patterns of a neuron as behaving like an audio signal, then LTP corresponds to pumping up the volume of particular firing patterns. But imagine an orchestra conductor who can only make the musicians play louder. Every symphony would be deafening by the time it ended! There has to be a way to turn the volume down as well as up. For LTP to be effective as a way to increase the strength of useful synapses, there would also have to be a process that can *decrease*

the strength of less useful synapses.

Soon after the discovery of LTP, such an opponent process was discovered ([Dunwiddie & Lynch, 1978](#)). **Long-term depression (LTD)**, also referred to as *synaptic depression*, occurs when synaptic transmission becomes *less* effective as a result of recent activity. One situation in which this happens is if presynaptic neurons are repeatedly active but the postsynaptic neurons do not respond. Neurons that fire together wire together, but connections between neurons that do not fire together become weaker, a change that is believed to reflect a weakening in synapses. As with the synaptic changes in LTP, researchers have various ideas about how the weakening might occur: there may be a decrease in the responsiveness of postsynaptic neurons, a decrease in neurotransmitter release by presynaptic neurons, or long-term structural changes in the neurons and synapses. As with LTP, many of the details of LTD remain to be worked out. Scientists still have a long way to go to understand synaptic plasticity and its relationship to learning and memory.

long-term depression (LTD)

A process in which synaptic transmission becomes less effective as a result of recent activity; with long-term potentiation, LTD is widely believed to represent a form of synaptic plasticity that could be the neural mechanism for learning.

TEST YOUR KNOWLEDGE

Synaptic Plasticity

Synaptic plasticity is one of the most researched phenomena in the field of neuroscience, yet many of its features remain poorly understood. Identify which, if any, of the following statements accurately describe(s) what is known about synaptic plasticity. ([Answers appear at the end of the chapter.](#))

1. Synaptic change can be produced through electrical stimulation.
2. Whenever firing patterns change in a neural circuit, synaptic change has occurred somewhere in the circuit.
3. Synaptic plasticity can weaken or strengthen connections between neurons.
4. Synaptic plasticity can be measured in humans using fMRI.
5. LTP is observed only in animals that have recently been learning.

How Researchers Change Brain Activity

LTP can occur naturally, but its original discovery was anything but natural. When Lømo electrically stimulated hippocampal axons in rabbits, he *really* stimulated them—think thousands of neurons simultaneously playing a drum roll of action potentials. This sort of extreme,

artificially induced firing is what made Lømo's discovery possible. If he had simply recorded hippocampal neurons going about their normal business, he never would have discovered LTP. Being able to affect what neurons do has proven to be critical not only to understanding how engrams may be stored but also to exploring the role that different brain regions play in learning and memory processes.

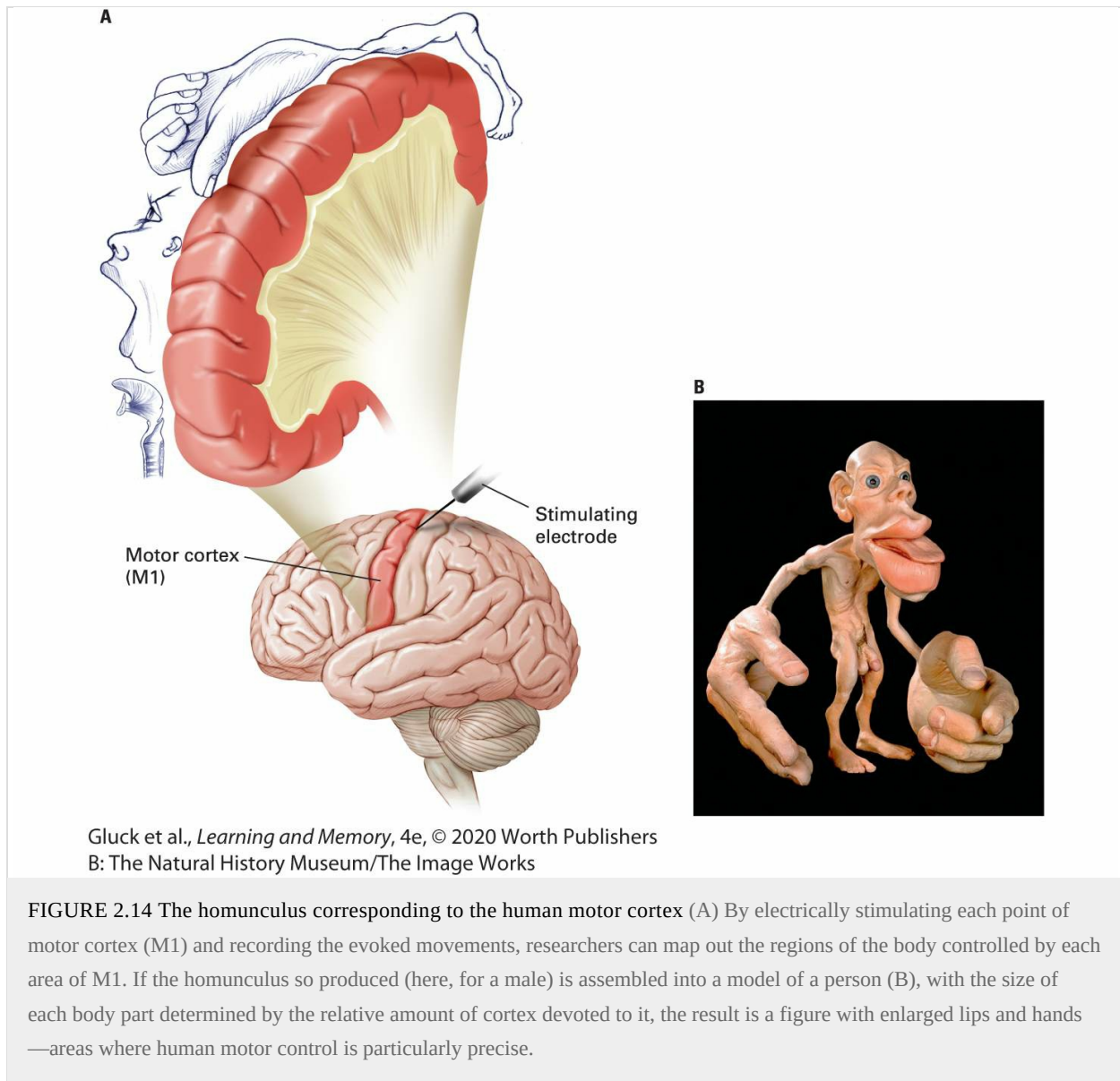
Electromagnetic Stimulation of Neurons

We noted earlier that in addition to using microelectrodes to monitor neural activity, researchers can also use microelectrodes to stimulate neural activity by delivering tiny amounts of electrical current into the brain. As you read in [Section 2.2](#), when neurons fire, an action potential sweeps down the axon, triggering the release of neurotransmitters into the synapse. Researchers can use stimulating electrodes to cause spiking activity to happen where and when they want it, as Lømo did in his studies of LTP.

Electrical stimulation of neurons was used as early as the 1800s, to prove that neural activity in the motor cortex produces motor behavior. Pavlov, for instance, was able to produce a wide range of movement patterns in an anesthetized dog by electrically stimulating its motor cortex ([Pavlov, 1927](#)). Similar techniques can be used in primates to map which parts of the motor cortex are responsible for generating movements in particular body parts. For example, electrical stimulation delivered to certain neurons in M1 in the right hemisphere, near the top of the brain, cause a monkey's lips to twitch. A little farther down, and an arm might twitch. Still lower, and movements occur in the legs.

By painstakingly testing the effects of stimulating each point in M1, scientists can draw a map—called a *homunculus* (or “little man”)—on the surface of M1, showing which parts of the body each subsection of M1 controls. The homunculus for M1 in humans ([Figure 2.14A](#)) has been worked out with the assistance of patients who were candidates for brain surgery (for example, to remove a tumor). Before removing any brain tissue, neurosurgeons do preliminary testing, which often involves cutting away a piece of the skull to expose the brain underneath and then carefully stimulating different areas. The idea is to determine whether the brain tissue can be cut away without leaving the patient in worse shape than before. To remove a tumor, for instance, it may be reasonable to risk damaging the part of M1 that controls movements in one leg, but risk to other parts—say, the areas that control the tongue and allow swallowing and speaking—may call

for extra caution.



Description

Section A shows an external view of the brain with a stimulating electrode attached to the Motor cortex (M1). A magnified view of the motor cortex is shown alongside a pencil drawing of an elongated arm and face.

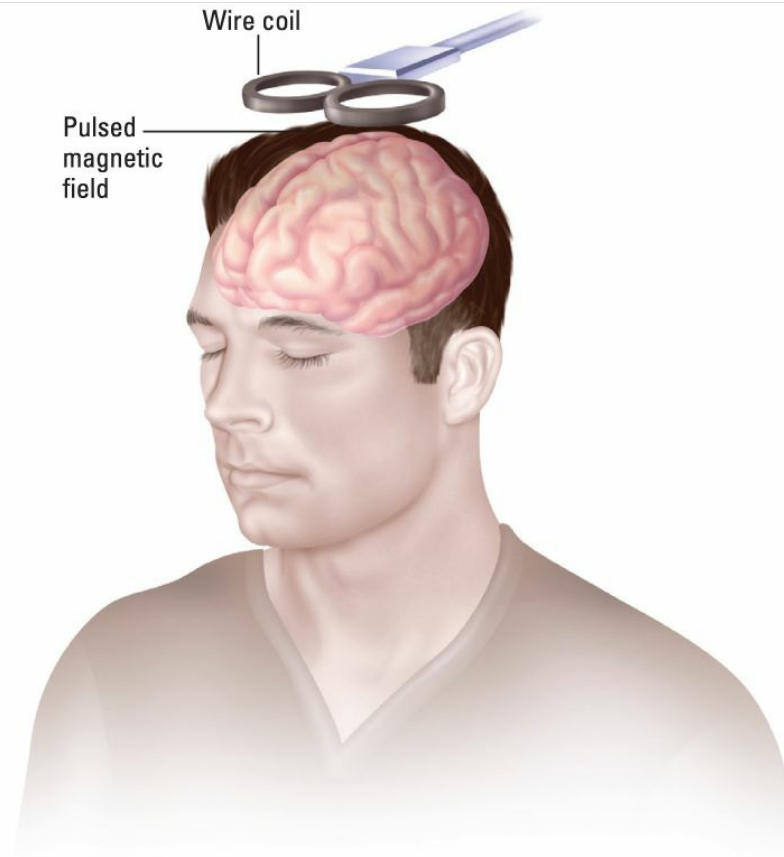
Section B shows a figure of a male, with the size of his body parts exaggerated.

Looking at the homunculus of [Figure 2.14A](#), you will notice that some body parts (the lips and hands, for example) seem grossly enlarged, while others (the arms and legs) seem shrunken. In

other words, the physical size of a body area does not directly correspond to its relative size in the cortical map. In fact, if the homunculus were assembled into a figurine, it would look something like [Figure 2.14B](#). The distortions are not random. The parts of the body that are exaggerated on the homunculus are precisely those parts in which humans have the highest degree of fine motor control: fingers that are able to type, knit, and play the piano; lips and tongue that move through the complicated contortions of speech; and facial muscles that display emotion. Other areas of the body that are physically larger, like the arms and legs, have proportionately less fine motor control, and so proportionately less area of motor cortex is devoted to them.

Electrical stimulation can be used not only to generate movements but also to generate visual, auditory, and somatosensory sensations (by stimulating neurons in sensory cortices). It is also possible to evoke feelings of *déjà vu*, the illusion of feeling that a novel experience has happened before, by stimulating neurons within the temporal lobe. It is even possible to classically condition animals (as described in [Chapter 1](#)) by using one electrode to generate neural firing patterns that would occur during the sensation of a sound and pairing that with stimulation from a second electrode that provokes a reflexive motor response. This kind of “virtual reality” training is described in greater detail in [Chapter 4](#).

Neural stimulation studies in patients and animal models have greatly increased our understanding of how neural activity is translated into behavior. The relatively new methods of *transcranial magnetic stimulation (TMS)* and *transcranial direct-current stimulation (tDCS)* now allow researchers to extend these kinds of studies to humans not undergoing brain surgery. TMS changes activity in the cerebral cortex by generating strong magnetic pulses over the skull ([Figure 2.15](#)), and tDCS delivers low-level electrical current through electrodes placed on the scalp.



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

FIGURE 2.15 Using TMS to modulate cortical activity This technique enables researchers (1) to disrupt cortical activity in volunteers to temporarily simulate cortical lesions or, conversely, (2) to temporarily increase the probability that cortical neurons will respond to inputs, thereby enhancing some cortical functions.

Description

The regions of the brain are highlighted within his head. A pulsed magnetic field is labeled on his head which is connected to a wire coil.

Both TMS and tDCS affect large cortical networks rather than individual neurons. Depending on the level of stimulation, transcranial stimulation can either facilitate neural functions or disrupt them. Some research suggests that both TMS and tDCS can improve function in patients with memory disorders ([Floel, 2014](#); [Reis et al., 2008](#)). Data from transcranial stimulation studies in humans may be most useful when combined with results from other studies of neural stimulation in animals and from functional neuroimaging studies in humans to help build the most complete

picture possible of which parts of the brain give rise to which kinds of behavioral changes.

Biochemical Control of Brain States

In addition to electrical and magnetic stimulation, a third method for manipulating neural activity is the use of **drugs**, chemical substances that alter the biochemical functioning of the body. For example, memory researchers have used the drug scopolamine to temporarily impair memory abilities by disrupting the actions of acetylcholine in the brain. Unlike electromagnetic brain stimulation, in which the main factors that determine effects are the amount and location of stimulation, neurochemical effects on the brain depend on a host of complex factors that are still being identified. Researchers may not always have a clear idea of why specific drugs enhance or hinder behavior, but there is no question that drugs can change neural activity, so it is no surprise that they can alter learning and memory processes.

drug

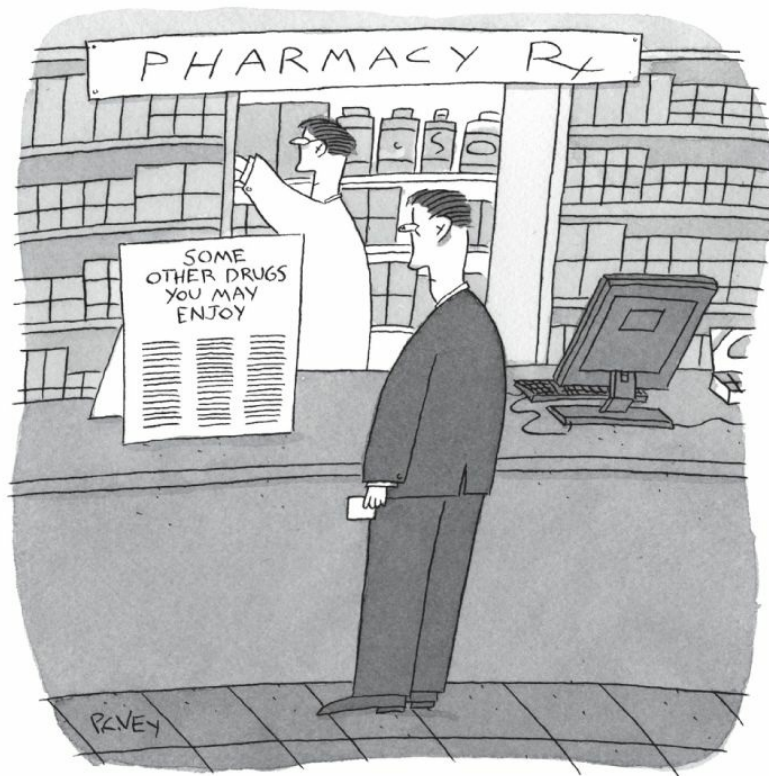
A chemical substance that alters the biochemical functioning of the body and, in many cases, affects the brain.

Drugs that interact with the brain generally do so by altering synaptic transmission. The effects of a drug on behavior depend on which neurotransmitters and neural circuits are involved and whether synaptic transmission is enhanced or impaired. Drugs can affect any of the four major processes of synaptic transmission described earlier: neurotransmitter release, activation of postsynaptic receptors, neurotransmitter inactivation, and neurotransmitter reuptake (depicted in [Figure 2.12B](#)).

Drugs can increase or decrease the ability of the presynaptic neuron to produce or release neurotransmitter (number 1 in [Figure 2.12B](#)). For example, amphetamine drugs alter the function of neurons that produce the neurotransmitter dopamine, causing the neurons to release greater than normal quantities of dopamine. This means that postsynaptic neurons receive stronger and more frequent messages than normal. Because the dopamine system is involved in the processing of reward in the brain, the result may be feelings of pleasurable anticipation or excitement. (There is more discussion of dopamine in [Chapter 5](#), on operant conditioning.)

Drugs can also increase or decrease the ability of postsynaptic receptors to receive the chemical message (number 2 in [Figure 2.12B](#)). For instance, the opiate drugs heroin and morphine are chemically very similar to a class of naturally occurring neurotransmitters called *endogenous*

opioids (also discussed further in [Chapter 5](#)). When heroin or morphine is released into the brain, molecules of the drug can trigger the receptors normally activated by the endogenous opioids. In effect, the drugs “fool” the postsynaptic neuron into thinking that strong signals are being received from many presynaptic neurons. As a result, weak chemical messages that would not normally cause firing in postsynaptic neurons instead cause lots of neurons to fire. The endogenous opioids seem to be important in how the brain processes and signals pleasure, which may explain why drugs that mimic endogenous opioids often cause intense feelings of pleasure.



Peter C. Vey/The New Yorker Collection/The Cartoon Bank

Drugs that work on the brain generally do so by altering what?

Drugs can alter the mechanisms for clearing neurotransmitter molecules out of the synapse (numbers 3 and 4 in [Figure 2.12B](#)). Some antidepressant medications (including the selective serotonin reuptake inhibitors, or SSRIs) work by reducing the rate at which serotonin is cleared from synapses. Thus, each time a presynaptic neuron releases serotonin molecules into the synapse, the molecules remain in the synapse longer, increasing their chance of activating a receptor and eliciting a reaction in the postsynaptic cell.

These are just some of the ways in which drugs can affect brain function. Commonly used drugs, including alcohol and nicotine, have been intensively studied. Still, their effects on neurons and synaptic transmission are so varied that the precise mechanisms by which these drugs affect the neural substrates of learning and memory are not yet entirely clear.

Few drugs have been developed specifically to affect learning and memory abilities (see “[Learning and Memory in Everyday Life](#)” for efforts in this direction). More commonly, a drug’s positive or negative effects on memory are considered side effects. For example, some types of general anesthesia administered to ease the pain of childbirth can also inadvertently “erase” a mother’s memory of her baby being born. Nevertheless, drugs are often used in learning and memory research to shut down processing in neural circuits, disrupt structural plasticity mechanisms (especially in studies of LTP), and of course to test their effects on diseases that impair learning and memory abilities.

LEARNING AND MEMORY IN EVERYDAY LIFE

Can a Pill Improve Your Memory?

If you have ever studied for a difficult exam, you have probably wished for a pill that could make your brain function like a copy machine. Instead of reading, reviewing, and rehearsing, you could swallow the pill, read the material once, and have the information encoded in your brain forever (or at least until the exam is over). Sounds like science fiction, right?

In fact, several companies, ranging from pharmaceutical giants to smaller biotech firms, are looking for a drug to improve memory in healthy people ([Lynch, Palmer, & Gall, 2011](#); [Stern & Alberini, 2013](#)). Some possible candidates are currently being tested on laboratory rats, and a few are even being tested in small groups of human volunteers. It remains to be seen which, if any, of these new drugs will be safe and effective.

Until a new generation of memory-boosting drugs becomes available, researchers continue to examine existing drugs, many already approved for the treatment of other illnesses, to see whether any might provide a memory boost in normal, healthy people. For example, several drugs used in treating Alzheimer’s disease—including donepezil (Aricept)—increase brain levels of the neurotransmitter acetylcholine, which is abnormally low in people with Alzheimer’s. These drugs can produce modest, temporary memory improvements in Alzheimer’s patients, raising the possibility that they might also improve memory in healthy (or mildly impaired) adults ([Whitehead et al., 2004](#)). However, there is little evidence so far to suggest that the drugs can boost memory in otherwise healthy people ([Stern & Alberini, 2013](#)).

Another approach to finding memory-enhancing drugs is based on the fact that attention and concentration can increase the storage and retention of new memories. Perhaps drugs that improve attention will also improve memory. Such attention-boosting drugs include modafinil (Provigil), which is used to treat sleep disorders, and methylphenidate (Ritalin), used to treat attention-deficit/hyperactivity disorder (ADHD). Many college students already pop Ritalin in an effort to boost studying or exam performance. But it is not clear that boosting attention beyond normal levels is necessarily good for memory. The jury is still out on whether these drugs improve memory in healthy humans ([Lynch, Palmer, & Gall, 2011](#)). The bottom line is that, so far, no pill can substitute for the hard work of learning. Instead of spending money on “brain-

boosting” drugs of questionable efficacy and safety, healthy people are best advised to do their learning the old-fashioned way: by devoting the necessary time to study.

Interim Summary

- Accidental brain lesions in humans have revealed much about how different brain regions function. Intentional brain lesions in animal models have similarly provided insights into how different regions contribute to learning and memory.
- The neural transmission that enables stimuli to generate responses takes place across tiny gaps, or synapses: the presynaptic (or sending) neuron releases neurotransmitters into the synapse; these chemicals cross the synapse to activate receptors on the postsynaptic (or receiving) neuron.
- Long-term potentiation (LTP) occurs when synaptic transmission becomes more effective as a result of strong electrical stimulation of neurons.
- An opponent process to LTP, called long-term depression (LTD), occurs when synaptic transmission becomes less effective after neurons do not fire together.
- Researchers can use implanted electrodes to stimulate neurons into activity so that the sensations or responses they evoke can be observed. Just as lesions can degrade memory abilities, stimulation can sometimes enhance memory.
- Drugs are chemicals that alter the biochemical functioning of the body. Drugs that affect the brain generally change neural activity by interfering with synaptic transmission.

Synthesis

We have covered a lot of ground in this chapter. We started with the basic geography of nervous systems, moved on to some key principles of how the various brain regions process different kinds of inputs and outputs, and ended by looking at how synapses can change over time.

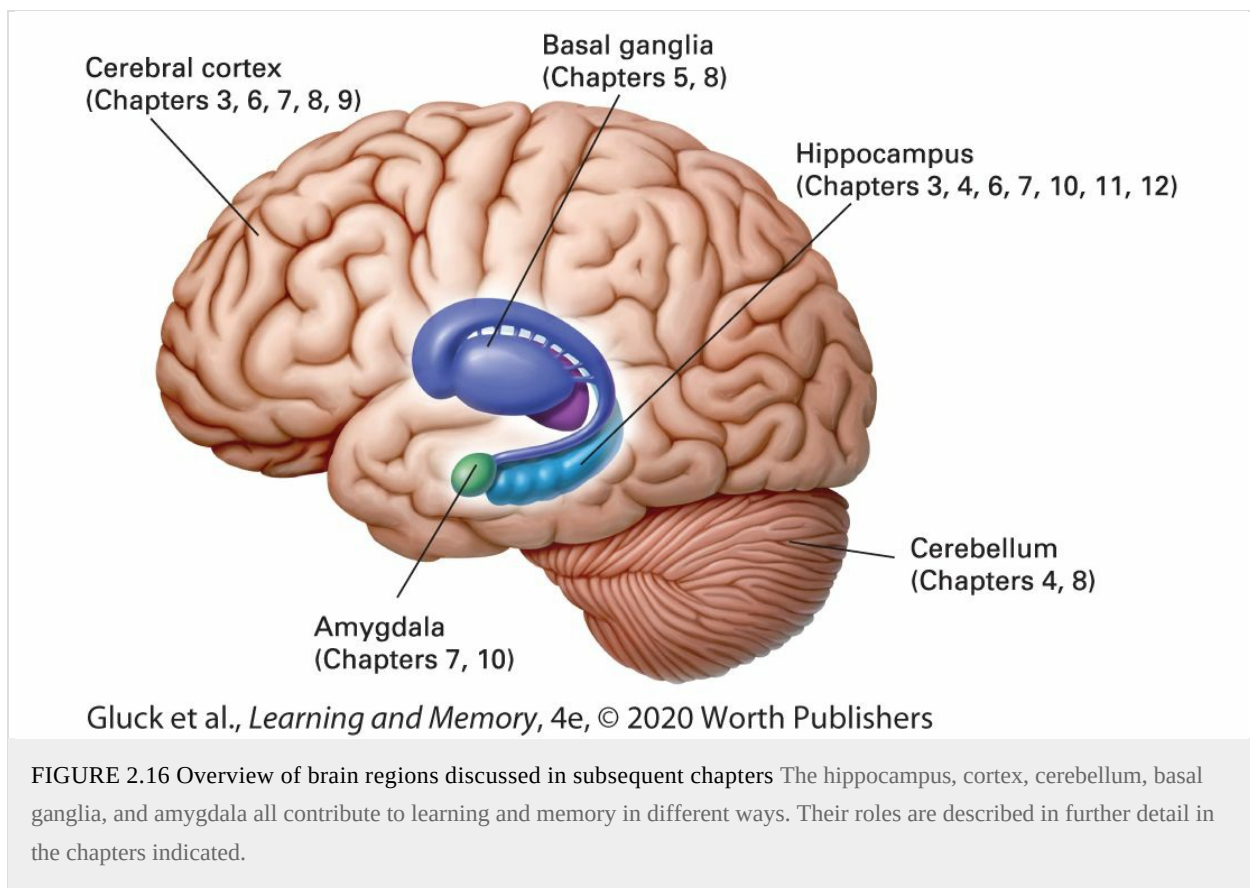
If you get the feeling that, for all this information, there are still a frustrating number of unresolved questions about the neural substrates of learning and memory, you are absolutely correct. But this is also a time when neuroscientists have access to an unprecedented selection of techniques: neuroimaging and recording methods that allow visualization of brain activity at multiple scales in living organisms; advanced microscopes that make synapses, dendrites, and neurotransmitter-containing vesicles visible; and systems capable of recording single-cell activity from hundreds of neurons simultaneously. These tools, now in fairly routine use, did not even exist a few decades ago.

In other chapters, we dig more deeply into the mechanisms that make learning and memory possible. The involvement of the hippocampus in these processes has been widely studied in humans and other animals. Consequently, this brain region is discussed in more chapters of this textbook than any other part of the brain. [Chapter 3](#) describes links between the hippocampus and spatial memories, [Chapter 4](#) discusses hippocampal contributions to eyeblink conditioning, and [Chapter 7](#) explores the dramatic impacts of hippocampal damage on memories for facts and events. In addition, hippocampal processing contributes to generalization of learning ([Chapter 6](#)), emotional learning ([Chapter 10](#)), and the social transfer of information ([Chapter 11](#)).

Intriguingly, the hippocampus is also one of the few regions in the mammalian brain where new neurons are born, a process that may contribute to our ability to learn new information early in life ([Chapter 12](#)).

Several regions of the cerebral cortex have also been shown to underlie various learning and memory capacities, so the discussion of cortical processing is likewise spread across multiple chapters. Changes in sensory and motor cortices associated with learning are described in [Chapters 3, 6, and 8](#), while the roles of the frontal and association cortices in memory formation and retrieval are reviewed in [Chapter 7](#). The frontal cortex also is discussed extensively in [Chapter 9](#), in relation to the ability to juggle thoughts and memories and to voluntarily manipulate how memories are used.

Three other brain regions also emphasized are the cerebellum, the basal ganglia, and the amygdala. The cerebellum has been studied extensively as a site of memory storage and processing in classical conditioning research ([Chapter 4](#)), as well as in relation to skill learning ([Chapter 8](#)). The basal ganglia are implicated in skill learning, too, particularly when learning involves reinforcement ([Chapter 5](#)), and in sensory-guided actions ([Chapter 8](#)). Finally, the amygdala is central to emotional learning, affecting how we learn emotional responses and also how we form memories of emotional events ([Chapter 10](#)). [Figure 2.16](#) summarizes where each of these brain regions is discussed in the book.



Description

The labeled parts are as follows. Cerebral cortex (Chapters 3, 6, 7, 8, 9), Basal ganglia (Chapters 5, 8), Thalamus, Hippocampus (Chapters 3, 4, 6, 7, 10, 11, 12), Cerebellum (Chapters 4, 8,) and Amygdala (Chapter 7, 10). The Cerebral cortex lies in the frontal brain. The basal ganglia are deep inside the cerebral hemispheres of the brain. The hippocampus is located in the temporal lobes of the brain. It has a horseshoe-shaped structure. The cerebellum is located in the hindbrain, adjacent to the brain stem. The Amygdala is located deep in the medial temporal lobe. It has an almond-shaped structure.

We hope that reading these chapters will engage all these regions of your brain and more, enabling you to better encode and organize your own knowledge about learning and memory phenomena.

Chapter 2 Review

Know Your Key Terms

axon

brainstem

cell body

central nervous system (CNS)

cerebellum

cerebral cortex

dendrite

diffusion tensor imaging (DTI)

drug

electroencephalography (EEG)

engram

enriched environment

event-related potential (ERP)

frontal lobe

functional magnetic resonance imaging (fMRI)

functional neuroimaging

glia

Hebbian learning

lesion

long-term depression (LTD)

long-term potentiation (LTP)

magnetic resonance imaging (MRI)

nervous system

neural connectivity

neuromodulator

neuron

neurophysiology

neuropsychology

neuroscience

neurotransmitter

occipital lobe

[parietal lobe](#)
[peripheral nervous system \(PNS\)](#)
[phrenology](#)
[plasticity](#)
[postsynaptic](#)
[presynaptic](#)
[receptor](#)
[reflex](#)
[single-cell recording](#)
[structural neuroimaging](#)
[synapse](#)
[synaptic plasticity](#)
[temporal lobe](#)
[theory of equipotentiality](#)

Quiz Yourself

1. The brain and spinal cord together make up the _____. ([p. 37](#))
2. Sensory receptors in your fingers and toes are part of your _____. ([p. 37](#))
3. The temporal lobe, parietal lobe, occipital lobe, and frontal lobe are all subdivisions of the _____. ([p. 38](#))
4. The _____ looks like a smaller brain hiding underneath the cerebral cortex and helps coordinate sensation and movement. ([p. 39](#))
5. _____ collect neurotransmitters released from a presynaptic neuron. ([p. 61](#))
6. Varieties of cells such as astrocytes and oligodendrocytes that are found throughout the brain are collectively called _____. ([pp. 41–42](#))
7. A major technique currently used to collect structural images of human brains is _____. ([p. 40](#))
8. The fact that by nature babies placed underwater do not inhale water is an example of

- a(n) _____. (p. 47)
9. Neurons in the _____ play an important role in the generation and control of motor responses in humans. (p. 49)
 10. The prototypical connections between neurons are between _____ and _____. (p. 41)
 11. A(n) _____ is a hypothetical physical change in neurons that forms the basis of a memory. (p. 59)
 12. If electrodes are attached to a person's head, it is probably because _____ recordings are being collected. (p. 52)
 13. One method for activating neurons in the cerebral cortex of humans without requiring surgery is _____. (p. 65)
 14. Chemical substances that, when brought into the body, can change how long neurotransmitters can activate receptors in a synapse are called _____. (p. 68)
 15. When connected neurons are firing at the same time, _____ can result. (p. 45)

Concept Check

1. In addition to learning to salivate whenever they heard a bell, some of Pavlov's dogs learned to salivate whenever Pavlov walked into the room. Use the concepts of synaptic plasticity and Hebbian learning to explain why this might have occurred. What region(s) of a dog's cortex might have changed as a result of this learning?
2. Neuroimages (such as fMRI brain scans) of different individuals performing the same task often differ greatly in the brain regions shown to be activated. Does this mean that the brains of these individuals function differently? If not, why not?
3. Drugs that block LTP in the hippocampus impair learning in some tasks but facilitate learning in other tasks. Similarly, some researchers have correlated LTP-like effects with learning in a variety of tasks, whereas others have observed learning in the

absence of these LTP effects. What does this tell us about the relationship between LTP and learning?

4. Carbon monoxide poisoning can damage many different parts of the brain, resulting in many different kinds of deficits—for example, severe impairments in language or an inability to recognize objects. Describe one way a neuropsychologist could determine what part(s) of the brain might have been damaged.
5. Lashley's findings from lesion experiments in rats suggest that the brain can function when only part of the cerebral cortex is available. In addition, invertebrates have been learning successfully for millions of years with very different sorts of nervous systems and less than 1% of the total neurons mammals have. What does this information imply about the role of the cerebral cortex in learning and memory?

Answers to Test Your Knowledge

Equipotentiality Versus Phrenology

Gall proposed that particular mental abilities are associated with specific cortical regions. Lashley proposed that mental abilities are not associated with specific cortical regions but instead can be performed by any sufficiently large region of cortex. Gall used correlations between skull features and individual variations in humans to support his ideas. Lashley used differences in the capacity of rats to learn mazes after undergoing cortical lesion to support his theory.

Synaptic Transmission

All of the statements correctly describe processes of synaptic transmission.

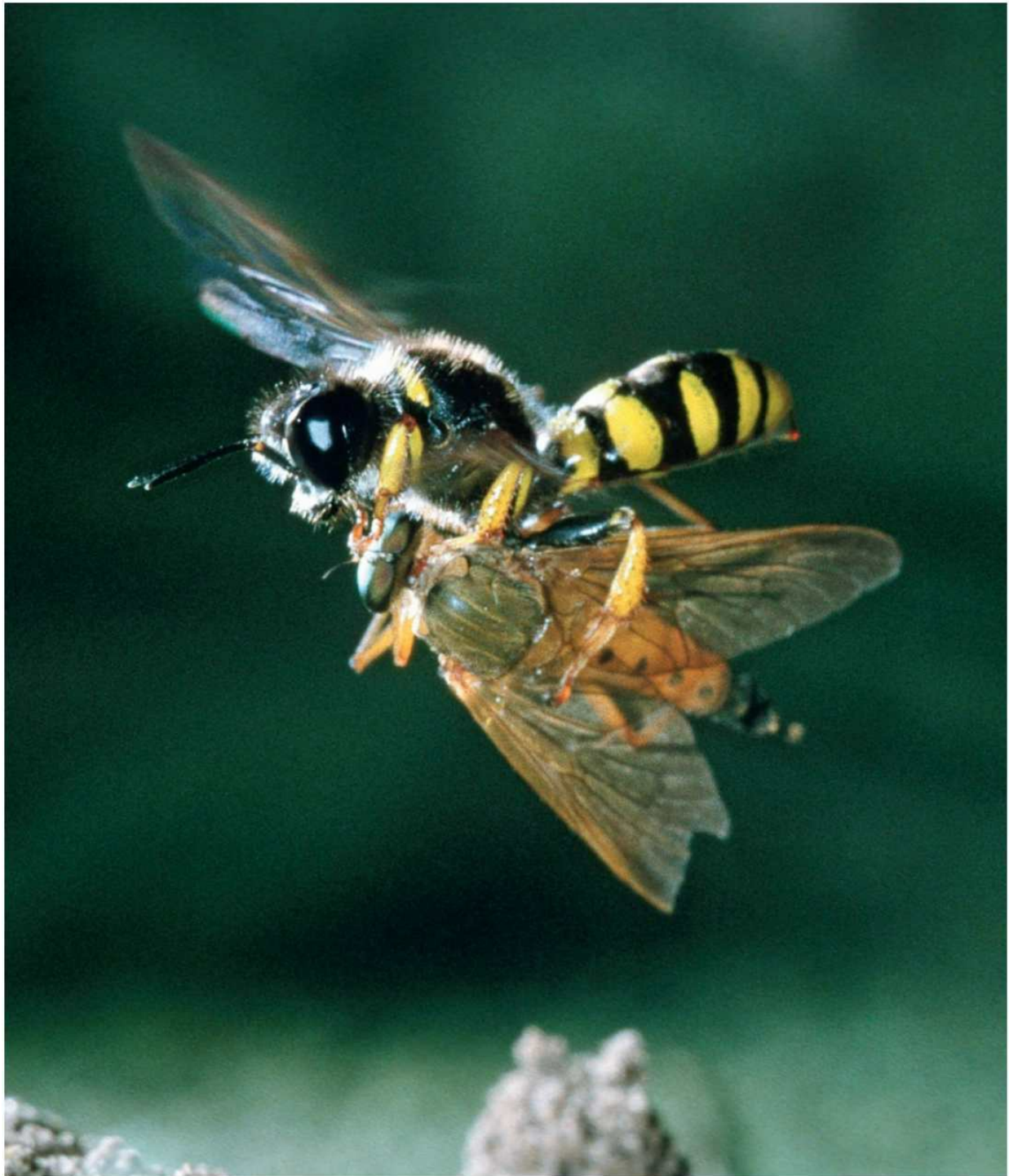
Synaptic Plasticity

1. True. This is what happened in the original LTP studies: the researchers stimulated one neuron and observed LTP in the postsynaptic neuron, indicating that the synapse had been strengthened.

2. False. A neuron changes its firing pattern when its inputs change, whether or not any synaptic change takes place.
3. True.
4. False. fMRI does not measure neural activity and cannot detect synapses; it measures changes in blood oxygenation, which is an indirect measure of neural activity.
5. False. LTP has only occasionally been observed in animals that are learning. It is commonly measured independent of any observable behavior.

Learning Module

Chapter 3 Habituation, Sensitization, and Familiarization Learning About Repeated Events



J Meul-Van Cauteren/Blickwinkel/AGE Fotostock

Behavioral Processes

[Recognizing and Responding to Repetition](#)

[*Learning and Memory in Everyday Life: Sex on the Beach*](#)

[The What and Where of Learning from Exposure](#)

[*Learning and Memory in Everyday Life: Unconscious Racial Bias*](#)

Brain Substrates

[An Invertebrate Model System](#)

[Perceptual Learning and Cortical Plasticity](#)

[Hippocampal Involvement in Spatial Learning and Familiarity](#)

Clinical Perspectives

[Rehabilitation After Stroke: Habituation Gone Awry](#)

[Sensitization to Stress in Anxiety and Depression](#)

[Human–Machine Interfaces: Regaining Sensory Modalities Through Perceptual Learning](#)

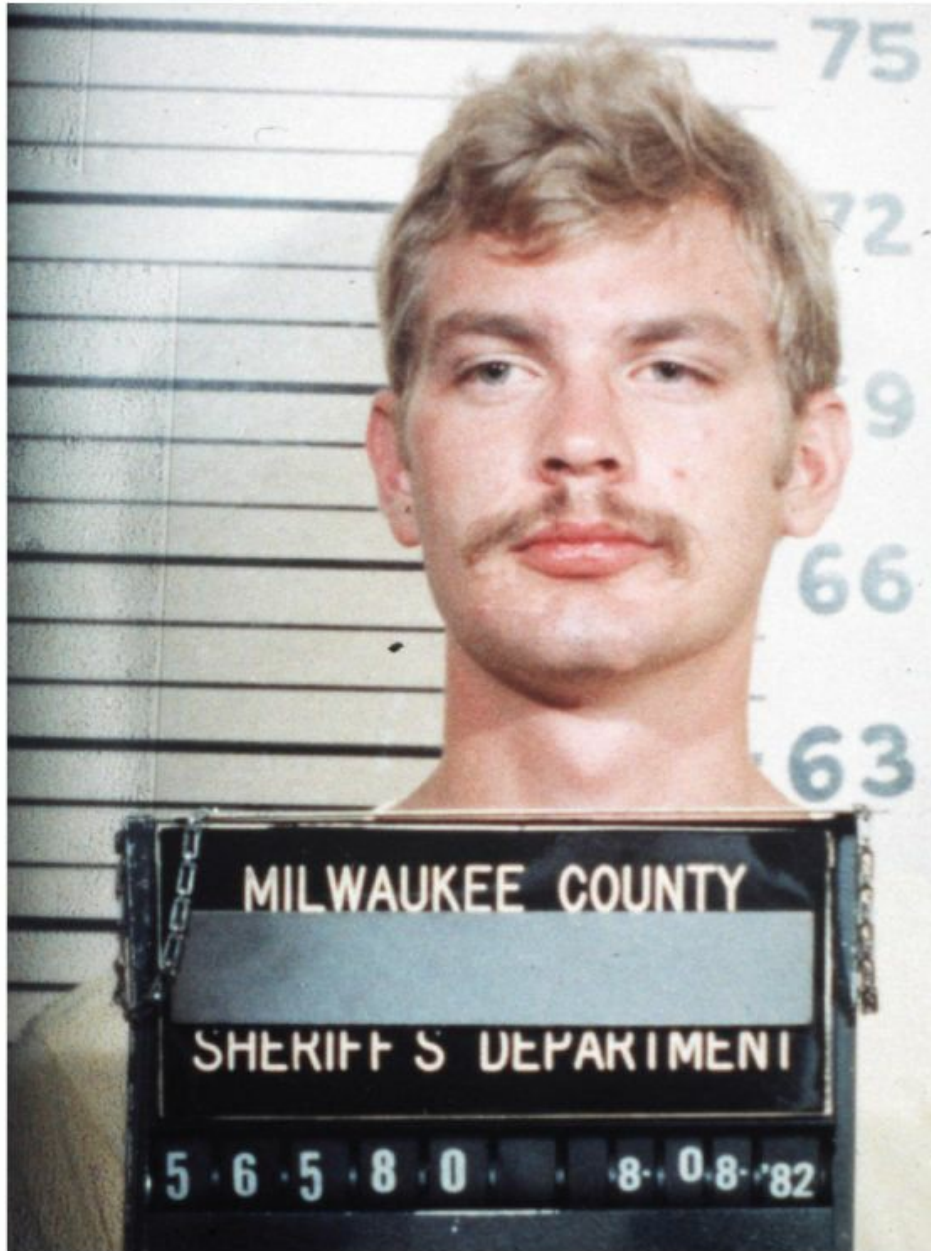
JEFFREY’S GRANDMOTHER WAS FED UP. It was 2 a.m., and once again her grandson was banging around in the basement. She couldn’t remember how many times she had told him to stop making such a racket. It had taken her a couple of years to get used to the neighbor’s dogs barking all night. They almost never woke her up now. But Jeffrey’s noisiness was another matter altogether. Every time he started up with the sawing, banging, and yelling, it seemed worse than the last time. She made up her mind to force Jeffrey to move out of the house. If she had known what he was doing to raise such a din, it would have disturbed her a lot more.

At first, Jeffrey Dahmer was annoyed at being kicked out of his grandmother’s house, but he soon got used to the convenience of having his own apartment. He took to cruising around the Pink Flamingo and other bars that were popular among young gay men, his potential victims. Dahmer had learned to recognize which of the customers were most likely to take his bait and follow him home. He couldn’t say what it was about them that let him know they were susceptible, but he was confident that he could discriminate the “maybes” from the “probably nots.”

During this same period, reports began to appear in the news media of young men going missing from the neighborhood. But disappearances were all too common in Milwaukee; for most people this was just more of the same. When one of Dahmer’s victims—a 14-year-old Laotian who spoke no English—escaped and was found running naked through the streets, police picked him

up and returned him to Dahmer's apartment. Dahmer convinced them that the boy was his lover and an adult and that they were just having a lover's tiff. The police noticed a terrible stench in the apartment, but noxious smells weren't uncommon in that part of the city. The officers left, and the boy was not seen alive again.

It wasn't until another victim escaped and flagged down a squad car that police returned to Dahmer's apartment. This time, they noticed some photos of dismembered bodies in the bedroom. That got the officers' attention, and when they investigated further, they found a human head in the refrigerator. The public, initially blasé about the news of one more captured criminal, paid considerably more attention when they learned that Dahmer was not only killing his victims but also eating them. The Jeffrey Dahmer case quickly became the biggest news story of its day.



AP Photo

In what ways did habituation in those around him help Jeffrey Dahmer evade notice and capture?

When an individual experiences the same event repeatedly, the brain accumulates memories of those experiences. Sometimes the memories lead a person to ignore additional repetitions of the events. Dahmer's grandmother heard the neighbor's dogs barking many times and eventually got used to them; the Milwaukee police had experienced many smelly apartments and consequently did not investigate the source of the putrid smell in Dahmer's apartment; the public was used to

news reports of disappearances and so did not think too much about what might be causing them. None of these occurrences elicited much of a reaction until new and more alarming aspects came to light.

Such decreased responding to repeated experiences is called *habituation*, and it is one of the most widespread forms of learning. All organisms ever tested—even those without a brain, such as protozoa—show habituation. It is just one example of how experiencing an event over and over can lead to learning about that event. Through habituation, a person learns to disregard an event; but in other instances, repetition can increase the attention given to the event. Jeffrey's grandmother became *sensitized* to the sounds she repeatedly heard coming from her basement, and eventually they annoyed her so much that she kicked Jeffrey out. Repeated experiences can also lead to *perceptual learning*, which enabled Jeffrey Dahmer to recognize suitable victims. This chapter focuses on the contrasting ways people and other organisms respond to repeated events, including how memories for repeated events are acquired.

3.1 Behavioral Processes

[Chapter 1](#) defines *learning* as a process by which experiences can lead to changes in behavior. Such changes in behavior are not always immediately obvious, however. For example, *latent learning*, described in the [Chapter 1](#) discussion of Edward Tolman, takes place without any initial indications of changes in behavior.

As a student reading this chapter, you are actively attempting to understand and remember the information it contains. At the same time, you are automatically forming memories of your reading experiences. As a student reading this chapter, you are actively attempting to understand and remember the information it contains. Did that last sentence seem familiar? Although you are probably not attempting to memorize every word of every sentence in this book, it is still relatively easy for you to recognize a sentence you have just read. To an outside observer, however, you are simply a person staring at words. There is no direct evidence that you understand the words you are looking at, much less that you have detected any repetition within the sentences. Your learning and recognition are effectively hidden from the world.

As a student reading this chapter, you are easily able to detect that repetition is occurring. As a student reading this chapter, you should be curious about how it is that you and other organisms do this. As a student reading this chapter, you should recognize that these are exactly the kinds of repetitions that might lead to habituation, if not for the variation in the way each repetition ends. As a student reading this chapter, you might also be aware of how this sort of persistent repetition can easily become aggravating. (If so, you are beginning to understand what sensitization is like.) As a student reading this chapter, you have just directly experienced some of the phenomena associated with latent learning about repeated events.

This section introduces you to learning processes that occur when you respond reflexively to repeated events, as when you habituate, and also to learning processes that occur when you repeatedly encounter similar stimuli while actively exploring the world around you, as when you recognize a celebrity in a magazine you are thumbing through.

Recognizing and Responding to Repetition

Suppose a man who was born and raised on the Caribbean island of Jamaica has never seen

snow. If he moves to Buffalo, New York, he will probably be excited and fascinated by his first snowfall. But a man who has grown up in Buffalo will react to the same snowfall very differently. For him, snow is recognizable, common, nothing to write home about—something he accepts and lives with on a daily basis.

Everything is novel the first time it happens to you. Even the most ordinary events become mundane only after repeated exposures. Through repetition, you may learn not to respond to a particular event, even if—like the man from Jamaica encountering snow—you originally responded with great excitement. This kind of learning, [habituation](#), is formally defined as a decrease in the strength or occurrence of a behavior after repeated exposure to the stimulus that produces the behavior. Habituation is sometimes described as the simplest or most basic kind of learning. Nevertheless, experimental studies conducted over the past century have yet to reveal exactly how habituation works ([Rankin et al., 2009](#); [Thompson, 2009](#)). In the following sections, we describe some of what researchers have discovered about habituation and its underlying mechanisms.

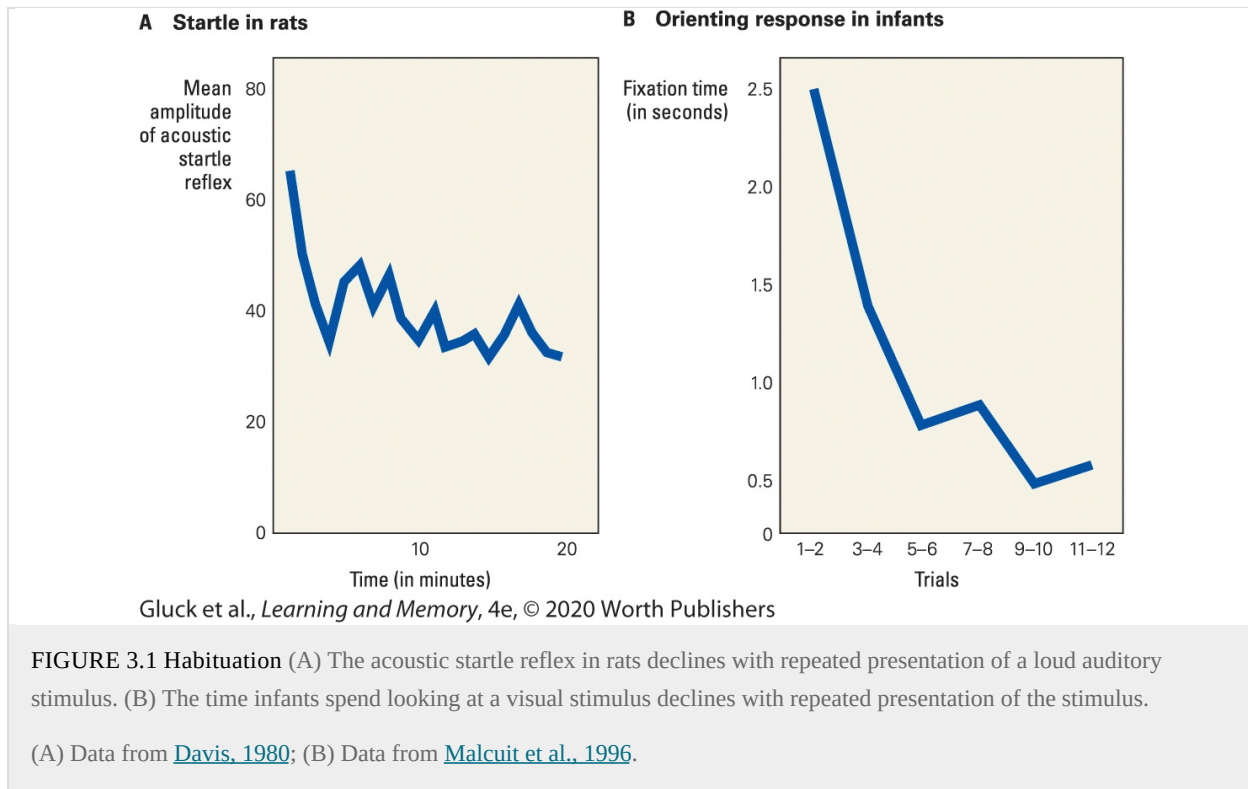
habituation

A decrease in the strength or occurrence of a behavior after repeated exposure to the stimulus that produces that behavior.

General Features of Habituation

If you have ever moved into a new home, you’ve experienced habituation. Possibly, the first night or two, you had trouble getting to sleep because of the strange noises outside your window (whether wailing police sirens or chirping crickets). But after a few nights, you probably were no longer awakened by the noises and slept until morning.

In the laboratory, researchers examine simpler examples of habituation that they can describe in terms of a single, easily controlled stimulus and a single, easily measurable response. One such response is the [acoustic startle reflex](#), which is a defensive response to a loud, unexpected noise. When a rat in an experimental chamber is startled by a loud noise, it jumps—much as you might jump if someone sneaked up behind you and yelled in your ear. If the same noise is presented to the rat over and over again, every minute or so, the rat’s startle reflex declines ([Figure 3.1A](#)), just as your responsiveness to noises would decrease after moving into a new home; if the process goes on long enough, the rat may cease to startle altogether. At this point, the rat’s startle reflex has habituated to the loud noise.



Description

The graph on the left is titled (A) Startle in rats. The horizontal axis shows Time in minutes. The vertical axis shows the Mean amplitude of acoustic startle reflex. The data in the graph are as follows. The curve starts from (2, 65) and then decreases to (4, 30). It then rises to (6, 45). Gradually, it goes up and down and reaches (20, 30).

The graph on the right is titled (B) Orienting response in infants. The horizontal axis shows Trials. The vertical axis shows Fixation time in seconds. The data in the graph are as follows. The curve starts from (1-2, 2.5), and abruptly declines down to (5-6, 0.8). Further, the curve transcends up and down the points and ends at (11-12, 0.6). All data are approximate.

acoustic startle reflex

A defensive response (such as jumping or freezing) to a startling stimulus (such as a loud noise).

Another common way to study habituation involves the [orienting response](#), an organism's natural reaction to sensing a novel stimulus or an important event. For example, if a checkerboard pattern (or any other unfamiliar visual stimulus) is presented to an infant, the infant's orienting response is to turn her head and look at it for a few seconds before shifting her gaze elsewhere. If the checkerboard is removed for 10 seconds and then redisplayed, the infant will respond again—but for a shorter time than on the first presentation ([Figure 3.1B](#)). The duration of staring, called the *fixation time*, decreases with repeated presentations of the stimulus,

in a manner very much like the habituation of a rat's startle reflex ([Malcuit, Bastien, & Pomerleau, 1996](#)).

orienting response

An organism's innate reaction to a novel stimulus.

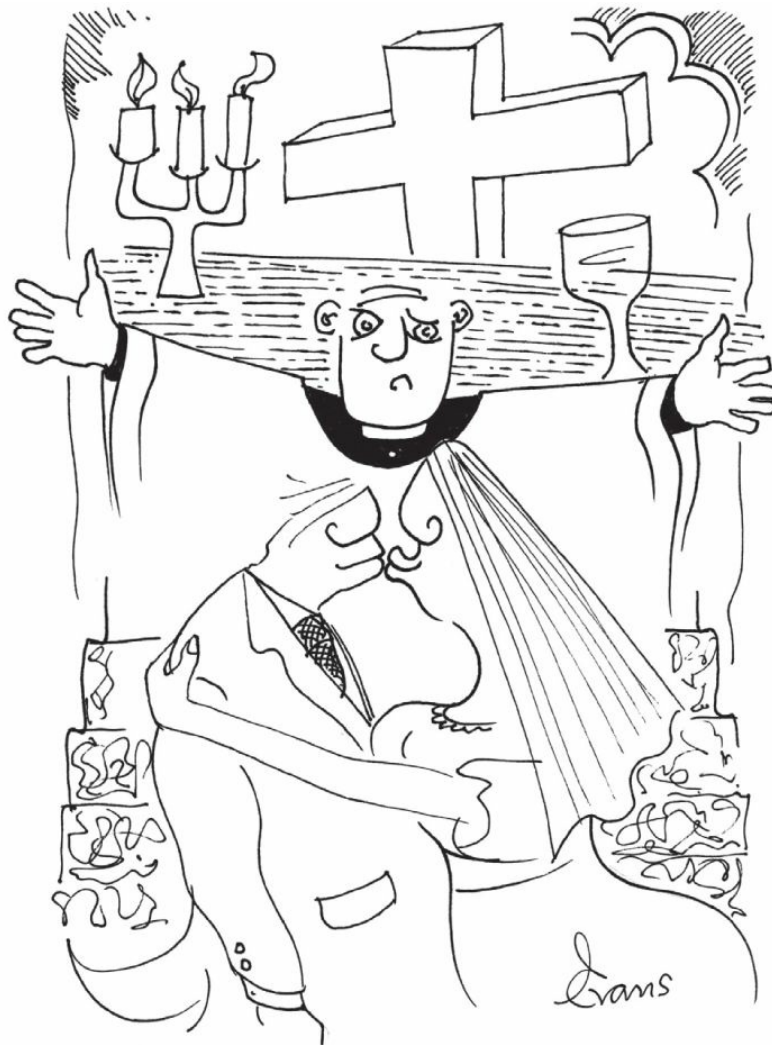
Normally, habituation is advantageous for an organism. Through habituation to familiar stimuli, the individual avoids wasting time and energy on unnecessary responses to each repeated event. But habituation carries risks. A deer that has gotten used to the sound of gunshots is a deer whose head may end up as a hunter's trophy. A poker player whose responses become habituated to the excitement of winning a small pot may start to play for larger and larger stakes, putting his finances at risk. The dangers of habituation are immortalized in the story of the boy who cried wolf. In this folk tale, the boy plays practical jokes on his neighbors, calling them to come save him from an imaginary wolf; eventually the villagers learn there is no reason to respond when he calls. Later, a real wolf attacks, but the villagers ignore the boy's cries, and no one comes to save him.

This might seem like a case in which the villagers just learned that the boy was unreliable. Think, however, about situations in which you have heard a fire or car alarm go off repeatedly for no apparent reason. Each time such an alarm goes off, you become more skeptical, and at the same time, your orienting response and startle reflex likely decrease. Any time your response decreases with repeated experiences, there is a good chance that habituation is occurring. Of course, it is also possible that you are simply falling asleep, especially if the alarm is going off at your bedtime. One way that researchers are able to distinguish habituation from fatigue and other causes of decreased behavioral responding to repetition is by disrupting the repetition of experienced events, as described next.

Stimulus Specificity and Dishabituation

An important feature of habituation is that habituation to one event does not cause habituation to every other stimulus in the same sensory modality. In other words, habituation is *stimulus specific* ([Thompson & Spencer, 1966](#)). When a baby's orienting response to one visual stimulus (say, a donut shape) has decreased after several repetitions, the baby will still show a strong orienting response to a new visual stimulus (say, a cross shape). This renewal of responding provides evidence of habituation to the first visual stimulus, because if the baby is simply falling

asleep, it should not matter what visual stimulus is presented.



“May the habituation commence.”

Andrew Evans/www.CartoonStock.com

Is habituation an inevitable consequence of long-term relationships? Read on ...

Habituation can occur even for highly arousing events. In the laboratory, a male rat will mate with an unfamiliar female many times over a period of a few hours but eventually reaches a point at which the mating stops. If the now-familiar female is replaced with a new female, however, the male rat will rush to mate some more. This recovery of the mating response shows that habituation occurs with the first partner and that the rat does not merely run out of energy or interest in sex ([Dewsbury, 1981](#); [Fisher, 1962](#)). (See “[Learning and Memory in Everyday Life](#)”

for information on habituation of human sexual responses.)

LEARNING AND MEMORY IN EVERYDAY LIFE

Sex on the Beach

Advertisements for travel to exotic locales with long, sandy beaches often show happy couples falling in love all over again, rediscovering the romance that may have drained out of their everyday existence back home. Can two people really reignite their old flame simply by visiting a new location? The answer is: probably—and the reason is related to how habituation works.

Most of the research on sexual attraction in humans has focused on arousal in male undergraduate volunteers viewing sexually explicit photos. Such studies have shown that if the same arousing photos are presented repeatedly, males respond less strongly ([Koukounas & Over, 2001](#); [Plaud, Gaither, Henderson, & Devitt, 1997](#)). Relatively few studies of habituation of sexual arousal have been conducted with women participants. One reason is that women usually do not become as aroused as their male counterparts when viewing sexually explicit photos. Obviously, it is hard for researchers to measure decreases in an arousal response if they can't reliably elicit arousal to begin with. But in studies that have managed to solve this problem, female undergraduates habituate to sexually arousing stimuli to a lesser degree than do male undergraduates ([Laan & Everaerd, 1995](#); [Youn, 2006](#)).

An interesting aspect of sexual habituation is that it seems to happen without conscious awareness. For example, male students in a sexual habituation experiment often show habituation within a single session, responding less and less to the same sexually explicit photo as the session goes on—but they also show habituation across sessions, responding less and less each day of a multi-day experiment ([Plaud et al., 1997](#)). Under these circumstances, participants often report that they were aware that their arousal was decreasing within a single session, but they seemed to be unaware that it also decreased across sessions. It may be that continuous but imperceptible decreases like this, affecting arousal at the sight of a long-term partner, are a factor in promiscuity and infidelity, posing a threat to stable relationships and perhaps also contributing to the spread of sexually transmitted infections ([Plaud et al., 1997](#)).

How can someone in a long-term relationship deal with the scourge of sexual habituation? Prolonged abstinence could lead to *spontaneous recovery* of interest. Another option is to introduce novel stimuli to bring about dishabituation. So the next time you are feeling bored with an old relationship, a trip to Tahiti might be just what the doctor ordered!

In some cases, presenting a novel stimulus can lead to recovery of responses after an individual has habituated to familiar stimuli. For example, a baby shown a donut shape many times may show little interest the twentieth time it is presented. If, however, the baby is briefly shown a live kitten after the nineteenth repetition of the donut, the baby is likely to respond to the twentieth presentation of a donut shape as if it were a novel image, showing a much longer fixation time than in the kitten-free scenario. This renewal of responding after a new stimulus has been presented is called **dishabituation**. Dishabituation often occurs when an arousing stimulus (like a kitten) is introduced into a sequence of otherwise monotonous repetitions, but it can also accompany less eventful changes. Simply adding motion to a familiar stimulus can lead to

dishabituation, as demonstrated when adults start waving toys around in front of an infant who seems uninterested. Dishabituation provides another useful way of demonstrating that the absence of responding to a repeated stimulus is indeed the result of habituation and not some other factor, such as fatigue.

dishabituation

A renewal of a response, previously habituated, that occurs when the organism is presented with a novel stimulus.

Factors Influencing the Rate and Duration of Habituation

How rapidly a response habituates and how long the decrease in responding lasts depend on several factors, including how startling the stimulus is, the number of times it is experienced, and the length of time between repeated exposures. It is relatively easy to get used to the feeling of the tag in the back of your shirt; most people can learn to ignore this stimulus relatively quickly. In fact, at this point, you probably would have to make an effort to notice that the tag is even there. It would probably take you longer to get used to the feeling of a spider crawling on the back of your neck—or you might never get used to that at all, even after many repeated experiences. In general, the less arousing an event is, the more rapidly a response to that event will habituate. Whenever habituation does occur, larger decreases in responding are seen after earlier repetitions than after later exposures (see [Figure 3.1](#)). In other words, the biggest changes in responding are seen when one is first becoming familiar with a stimulus. This pattern, or *learning curve*, is seen for many kinds of learning and is discussed in greater detail in [Chapters 4, 5, and 8](#).

Animals given sessions of multiple exposures to stimuli in which the exposures are separated by short intervals will typically show habituation after fewer exposures than will animals given sessions in which the same number of exposures are more spread out over time ([Rankin & Broster, 1992](#); [Thompson & Spencer, 1966](#)): more rapid repetition of a stimulus generally leads to more rapid habituation. Exposures that are repeated close together in time are called *massed*, whereas exposures that are spread out over time are called *spaced*. If your goal is to habituate your response to some repeated stimulus as rapidly as possible, your best bet is to find a way to make that stimulus as nonarousing as possible, to expose yourself to closely spaced repetitions of that stimulus, and to repeat the process frequently (as you do when you wear shirts with tags in the back).

The effects of habituation may last for a few minutes or several hours and, under some circumstances, may last a day or more. But they generally do not last forever and are especially likely to dissipate if the stimulus is absent for a while. Habituation that goes away in seconds or minutes is called *short-term habituation*; habituation that lasts longer is called *long-term habituation* ([Rankin et al., 2009](#)). If a rat has gotten used to a loud noise and then goes through a period of an hour or so in which the noise does not occur, the rat is likely to startle anew when the noise is played again, a process called **spontaneous recovery** (referred to in the box on [page 80](#)). In spontaneous recovery, a stimulus-evoked response that has been weakened by habituation increases in strength or reappears after a period of no stimulus presentation.

spontaneous recovery

Reappearance (or increase in strength) of a previously habituated response after a short period of no stimulus presentation.

The factors that determine how quickly an individual's response habituates also affect how long the effects of habituation last. Animals that experience massed exposures to a stimulus learn to ignore that stimulus faster than do animals given spaced exposures, but if they are retested after a relatively long break, the animals given massed exposures are also more likely to show spontaneous recovery. When exposures are spaced in time, it takes longer for responding to habituate, but once habituation occurs, it lasts for a longer time ([Gatchel, 1975](#); [Pedreira, Romano, Tomsic, Lozada, & Maldonado, 1998](#)).

This finding makes intuitive sense because animals that have gotten used to the intermittent occurrence of a stimulus should find the recurrence of the stimulus after a long interval to be familiar. As a student reading this chapter, you are easily able to detect that repetition is occurring, even when that repetition occurs after moderately long intervals. If your repeated experiences are spread out over time, the likelihood is greater that you will continue to recognize repeating events farther into the future. So, if you want habituation to last for as long as possible, your best bet is to repeatedly expose yourself to the relevant stimulus after longer and longer stretches of time.

Although spontaneous recovery might seem to suggest that habituation is a temporary effect, habituation effects accumulate over time, as in the following example of an infant's experiences with a donut shape. If an infant is shown a donut shape 20 times during a single session, her orienting response to that image will likely habituate. If a day or two later, the infant is shown

the donut shape again, spontaneous recovery will probably have occurred, and the infant's fixation time will be as long as if the image were completely novel. However, it may now take only 8 trials before the infant's orienting response becomes habituated: the effects of the repeated experiences have been effectively amplified by the prior repetitions. This shows that the effects of earlier repeated experiences have not simply faded away.

Furthermore, the mechanisms underlying habituation continue to change with repeated exposures, even when behavioral responses are no longer changing. For instance, a rat exposed many times to a loud sound might stop showing any indication that it even hears the sound; in this case, its response decreases to the point at which there no longer is a response. Nevertheless, if the sound continues to be repeated many times after this point is reached, the amount of time required before spontaneous recovery occurs will increase. In this case, the learning associated with repeated exposures is latent because there are no observable changes in the rat's behavior associated with the increased number of repetitions. The additional effects of repeated exposures after behavioral responding to a stimulus has ceased are evident only when subsequent tests show delayed spontaneous recovery ([Thompson & Spencer, 1966](#)).

General Features of Sensitization

Several times a year, news reports appear of a celebrity attacking a member of the paparazzi. The actor Sean Penn was charged with attempted murder after he grabbed a photographer by the ankles and held him over the side of a ninth-floor balcony. Rapper Kanye West was arrested for attacking a reporter at Los Angeles International Airport and destroying his camera. Given that celebrities have lots of photos taken of them by lots of people on a daily basis, you might expect that they would eventually become used to all the attention and take no notice of photographers. What causes some celebrities to become so aggressive when confronted with paparazzi? Is it just bad temper?

One possible explanation is that some celebrities have had negative experiences involving photographers in the past that are affecting their responses to new interactions with random members of the paparazzi. [Sensitization](#) is a phenomenon in which experiences with an arousing stimulus lead to stronger responses to a later stimulus. In some cases, a single, very intense stimulus can produce sensitization, whereas in others, repeated exposures are required. In some ways, sensitization seems to be the opposite of habituation. Whereas in habituation repeated

experiences can attenuate a rat's acoustic startle reflex, in sensitization repeated experiences can heighten it.

sensitization

A phenomenon in which a salient stimulus (such as an electric shock) temporarily increases the strength of responses to other stimuli.

No one is born liking hot sauce. Given the pain it can cause, most children avoid it. So how is it that many adults end up *liking* their mouth to burn? Maturation plays a part, but one of the best predictors of who will favor spicy foods is how often they are exposed to such foods ([Ludy & Mattes, 2012](#)). Spicy heat levels that are the norm in some cultures are intolerable in others, largely because repeated experiences with spicy foods can cause an increase in positive responses to those foods. There even is evidence that becoming sensitized to the taste of junk foods (Flamin' Hot Cheetos? Buffalo chicken wings?) may be contributing to the obesity epidemic in the United States ([Temple & Epstein, 2012](#)).



Vladimir Gjorgiev/Shutterstock

Some people acquire a preference for foods that are so spicy they produce a burning sensation. How might repeated exposures to a painful stimulus make it become more rewarding?

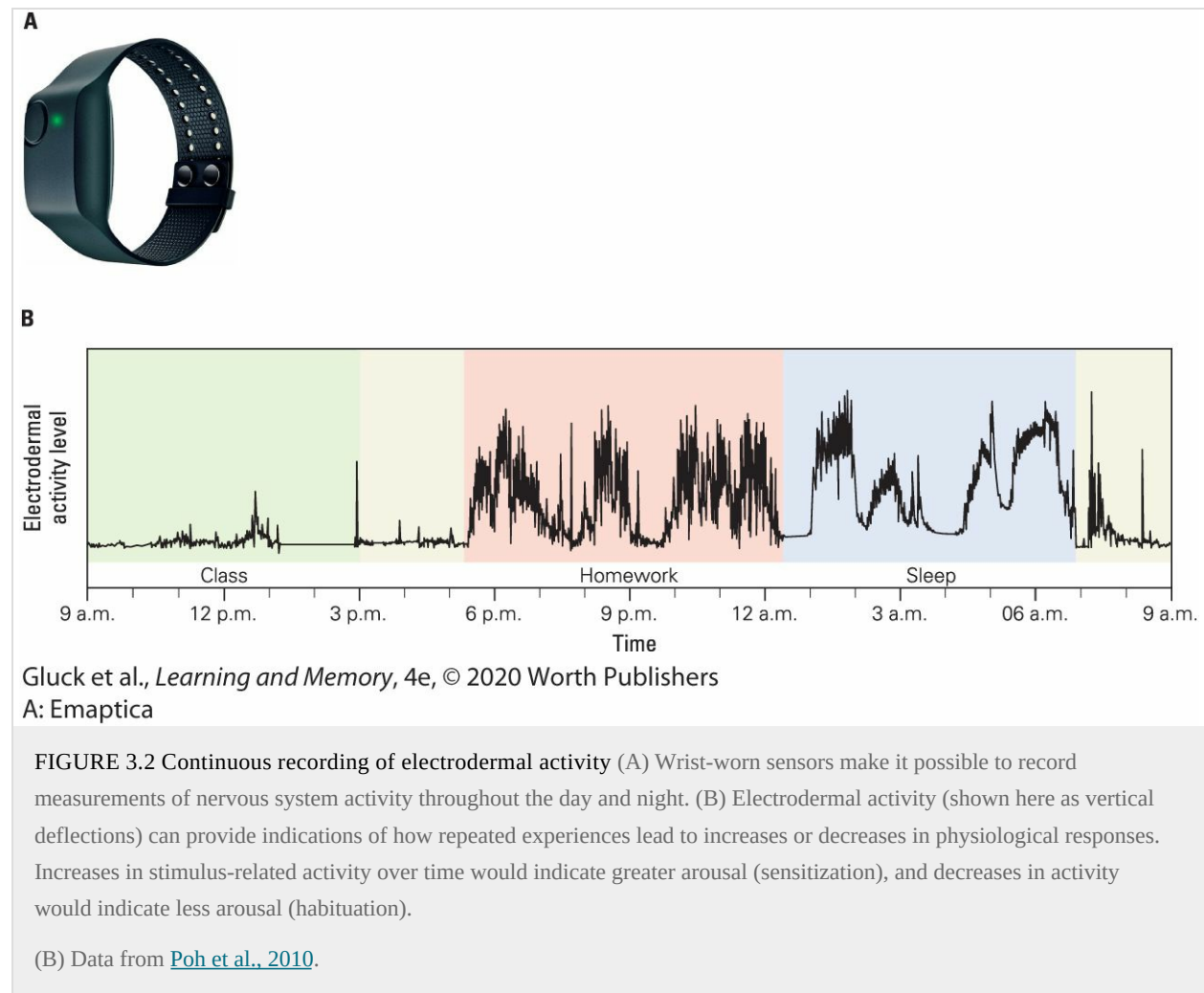
Like habituation, sensitization is seen in a wide range of species, including bullfrogs, sea slugs (as you will see in [Section 3.2](#)), and humans ([Bee, 2001](#); [Eisenstein, Eisenstein, & Bonheim, 1991](#); [Marcus, Nolen, Rankin, & Carew, 1988](#)). Also like habituation, sensitization can rapidly dissipate in some situations but can lead to longer-lasting learning in others—and as with habituation, the long-term effects can be difficult to predict ([Borszcz, Cranney, & Leaton, 1989](#); [Davis, 1972, 1980](#); [Poon & Young, 2006](#)). However, fewer exposures are typically necessary to produce sensitization than to produce habituation, and whereas habituation is stimulus specific, sensitization is not. For example, an animal's startle reflex may habituate to one loud tone that is repeated over and over; but if a different loud noise is presented, the startle reflex reappears in full force—habituation doesn't transfer to the new sound. By contrast, exposure to a sensitizing stimulus (such as an electric shock) can amplify the startle reflex to any stimulus that comes later: tone, loud noise, butterfly, or anything else. Similarly, a celebrity who catches a photographer peering into his house might be responding not just to that particular photographer but, as an aftereffect, to other even more annoying photographers that he has previously encountered.

One way that researchers can study sensitization experimentally in humans is by measuring [electrodermal activity \(EDA\)](#). EDA is a measure of changes in the skin's electrical conductivity that are caused by fluctuations in the activity of the peripheral nervous system. Emotional responses such as fear or surprise can produce significant fluctuations in a person's EDA, which is why measures of EDA are one of the main components of polygraphs, or lie detectors. In the laboratory, researchers record EDA using electrodes similar to those used for recording electroencephalograms (EEGs; see [Chapter 2](#) for details). Exposure to an unexpected loud noise (say, an explosion or a yell), which causes a pronounced acoustic startle reflex in humans, is also accompanied by a rapid, large change in EDA. A neutral musical tone may cause a mild orienting response as well as a small change in EDA. If the loud noise is played before presentation of the tone, the EDA change in response to the tone will be stronger than without the loud noise ([Lang, Davis, & Ohman, 2000](#)), showing sensitization.

electrodermal activity (EDA)

Fluctuations in the electrical properties of a person's skin that are a consequence of activity in the peripheral nervous system.

Portable sensors are now commercially available, making it possible for you to record your own EDA around the clock ([Figure 3.2A](#)). In principle, you could use such sensors to track and monitor events in your life that are leading to either sensitization or habituation ([Figure 3.2B](#); [Poh, Swenson, & Picard, 2010](#)). Access to such information might make it easier to avoid or mitigate some psychological disorders (discussed in more detail in [Section 3.3](#)).



Description

The graph labeled B has a horizontal axis which plots Time. It is divided into three parts as follows: Class, 9 a.m. to 3 p.m.; Homework, 3 p.m. to 12.30 a.m.; and Sleep, 12.30 a.m. to 7 a.m. The vertical axis plots Electrodermal activity level. The electrodermal activity is minimal during the class hours and maximum during the homework hours. The electrodermal activity is on an average during the sleep hours. A photo labeled A shows a wrist-worn sensor.

Loud noises can sensitize a person's response to tones, just as electric shock can sensitize an

acoustic startle reflex in rats. Surprisingly, if the order of events is reversed, the effects on responding can also reverse. Specifically, if a relatively quiet tone is presented just before a much louder and more startling noise, the startle reflex to the louder noise may be reduced relative to the response that would have occurred if only the startling sound had been heard. This effect is called *prepulse inhibition*, and it is similar to habituation in that some stimuli are “tuned out” on the basis of past experience, leading to reduced responses. However, prepulse inhibition is also similar to sensitization in that (1) the initial weak stimulus can affect responding to a wide range of subsequent stimuli, including stimuli in other modalities; and (2) a single presentation of the weak stimulus can produce the effect ([Braff, Geyer, & Swerdlow, 2001](#)).

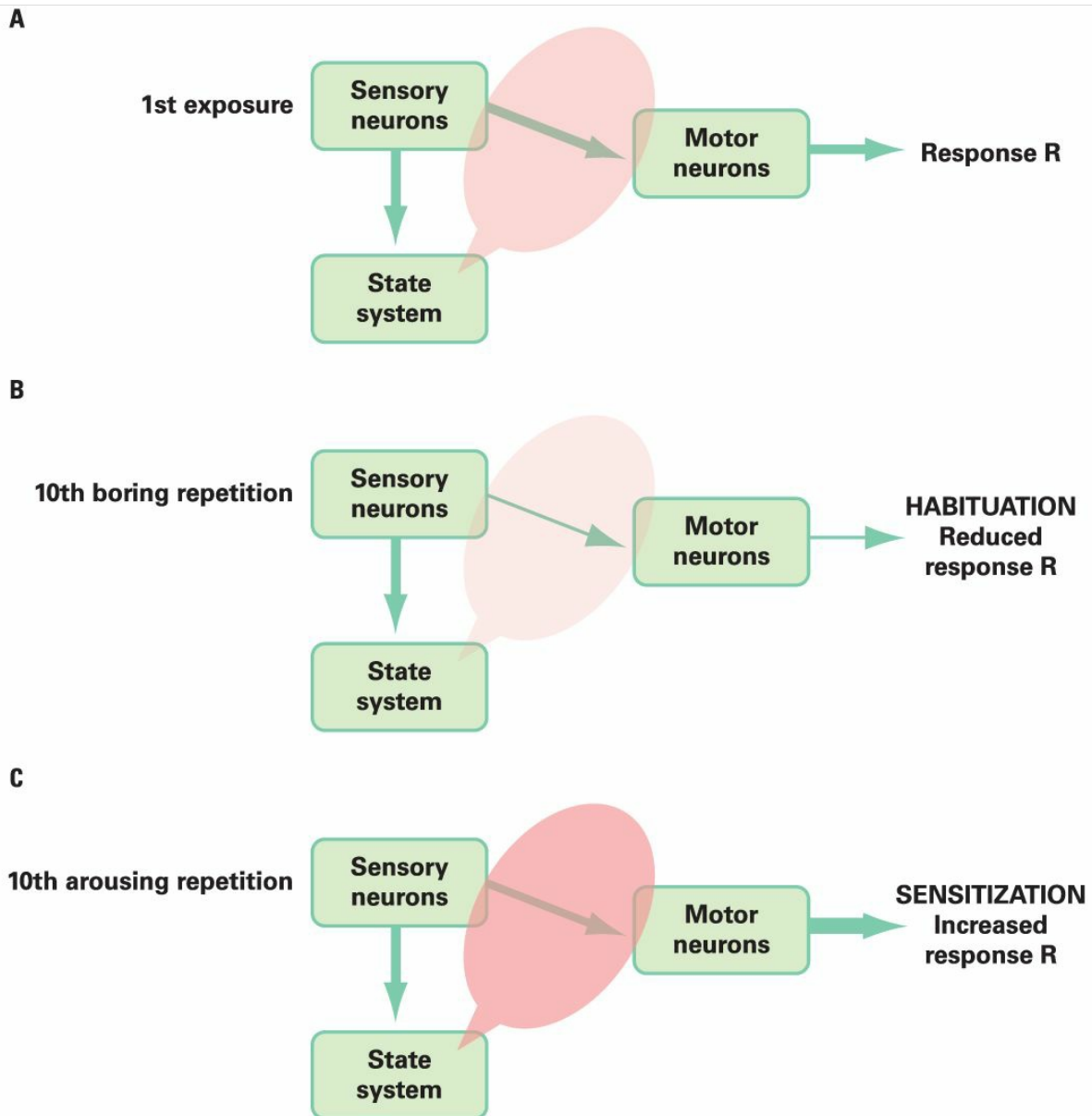
These properties have led some researchers to describe prepulse inhibition as a case of *desensitization*, the process in which past experiences reduce responses to a wide range of related stimuli ([Poon & Schmid, 2012](#); [Poon & Young, 2006](#)). You are probably familiar with the idea of people becoming “desensitized” to violence or to events that are unpleasant (like changing dirty diapers). It is difficult to distinguish desensitization from habituation in these natural contexts because both involve decreased responses. Laboratory studies of the neural processes engaged by habituation and desensitization provide new ways of disentangling these two closely related phenomena (discussed further in [Section 3.2](#)).

Interestingly, a series of events that might normally lead to habituation can, in a sick animal, lead to sensitization ([Domjan, 1977](#)). In other words, the state of the organism can play a large role in what it learns from repeated events. So, a celebrity who repeatedly encounters paparazzi while being sleep deprived, hung over, or ill may be more likely to become sensitized to annoying intrusions of privacy and therefore more likely to strike back than a healthier celebrity who is similarly beset by photographers.

Dual Process Theory

If the same repeated events can potentially lead to *either* habituation or sensitization, how can anyone predict what an organism will learn from repeated exposures? Novel stimuli are often arousing, but what is it that determines whether an event will generate increasing arousal with repetition as opposed to just becoming boring? One popular theory, called [dual process theory](#), suggests that, in fact, the effect of repeated events *is decided by the interplay of processes underlying sensitization and habituation* ([Groves & Thompson, 1970](#); [Thompson, 2009](#)). Here’s

how it works: imagine that when a stimulus S is presented, it evokes a chain of neural responses that (1) potentially leads to activation of a motor response R and (2) also activates a *state system* that increases arousal or excitability ([Figure 3.3A](#)). Habituation after repeated exposures to the stimulus (let's say 10 boring repetitions) can then be modeled as a weakening of the connection between S and R ([Figure 3.3B](#)) combined with only mild activation of the state system (producing moderate arousal). The weaker connection decreases the likelihood of activity within motor neurons, making the response to S weaker, or less likely to occur. If the stimulus is not very arousing, then the weakened connection essentially determines responding. Sensitization can then be modeled as an increase in the effect of the state system on sensory-evoked responses ([Figure 3.3C](#)), such that even harmless stimuli may be able to produce strong responses.



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FIGURE 3.3 The dual process theory of habituation Dual process theory suggests that both habituation and sensitization processes occur in parallel during every presentation of a stimulus and that the final response after repeated presentations results from the combination of both processes. (A) Initially, a stimulus such as S activates sensory neurons that lead to a motor response R and also activates a separate state system signaling detection of the stimulus. (B) In habituation, repeated “boring” presentations of S can weaken the connections between neurons (thinner arrows), thus reducing the strength of R or the likelihood that S leads to R. (C) In sensitization, repeated exposure to an arousing stimulus increases the likelihood that subsequent presentations of S lead to R by changing the global state of the circuit (pink balloons) such that all effects of stimuli are amplified.

(A) Information from [Groves & Thompson, 1970](#).

Description

The first illustration labeled A shows the first exposure. Three text boxes are given. The text box Sensory neurons branches in two: State system and Motor neurons. The text box Motor neurons branches into Response R.

The second illustration labeled B shows the tenth boring repetition. The data given in the illustration are as follows. Three text boxes are given. The text box Sensory neurons branches in two: State system and Motor neurons. The text box Motor neurons branches into HABITUATION Reduced response R.

The third illustration labeled C shows the tenth arousing repetition. The data given in the illustration are as follows. Three text boxes are given. The text box Sensory neurons branches in two: State system and Motor neurons. The text box Motor neurons branches into SENSITIZATION Increased response R.

dual process theory

The theory that habituation and sensitization are independent of each other but operate in parallel.

In dual process theory, both sensitization and habituation processes occur in response to every stimulus presentation, and it is the summed combination of these two independent processes that determines the strength of responding ([Groves & Thompson, 1970](#)). The actual outcome—the strength of the response to S on a given presentation—depends on such factors as how often S has been repeated and the intensity and recency of highly arousing events. It can also depend on whether other stimuli have activated the state system and on factors such as illnesses that might affect an organism's state. For stimuli that lead to little arousal, decreases in connection strengths will be the main determinants of how an organism's responses change over time, leading to the behavioral phenomenon that researchers call habituation. When stimuli are highly arousing, global effects tending to strengthen many connections will be more evident in responses, leading to the behavioral phenomenon known as sensitization. In dual process theory, both sensitization and habituation processes change over time such that the largest effects of repetition always occur in early exposures, producing the characteristic learning curves shown in [Figure 3.1](#).

Opponent Process Theory

Studies of emotional responses to extreme events—to a roller-coaster ride, for example—suggest that there are multiple phases of emotional responding. An initial phase that is scary is followed by a “rebound” effect of exhilaration. After repeated experiences, the initial fear responses may become weaker, whereas the rebound responses grow stronger (such that what was once scary can become fun). One model of this process, called *opponent process theory*, explains this effect as a way for organisms to maintain emotional stability.

Opponent process theory is similar to dual process theory in assuming that an experienced event leads to two independent processes—in this case, two emotional processes: one that is pleasurable and one that is less pleasant. The overall emotion a person experiences in response to an event is the combined result of these two independent processes. Repeated experiences have different effects on the initial reaction versus the rebound reaction, causing the initial response to habituate faster than the rebound. Thus, your first time skydiving may not be nearly as much fun as your fifth time because your initial response, a fear response, will be much stronger than the rebound “thrill.” Two features of the opponent process theory that distinguish it from the dual process theory are (1) the rebound, or opposing process, which is directly triggered by the initial emotional response, and (2) that both underlying processes in the opponent process theory are associated with emotional arousal (a change in the state of the individual).



Graiki/Moment/Getty Images

How does the opponent process theory explain why some people find jumping out of planes to be fun?

Both dual process theory and opponent process theory suggest that the learning that results from repeated experiences is not as simple as may first appear. A “simple” decrease or increase in responding to an increasingly familiar event may reflect multiple learning processes occurring in parallel. In addition, repeated experiences can change not only how an individual reflexively responds to familiar events but also how the person perceives and interprets those events, as described in the following section.

TEST YOUR KNOWLEDGE

Maximizing Habituation

In some situations—such as in a romantic relationship—it can make sense to try to minimize habituation. In contrast, if you discover that the shirt you are wearing is itchy, you’d be better off if you could maximize habituation and avoid sensitization. Try to come up with at least three strategies, aside from wearing a different shirt, to help yourself habituate to the repeated tactile stimulation produced by the shirt. (*Answers appear at the end of the chapter.*)

The What and Where of Learning from Exposure

Habituation and sensitization generally do not require much physical or mental effort or initiative. For example, babies can habituate to a tone in a laboratory simply by being there. Similarly, a couch potato may quickly learn to loathe certain annoying commercials simply by repeatedly sitting through them, without ever paying attention to the content. In many situations, however, individuals do exert some control over the kinds of stimuli they repeatedly experience. Animals rarely just sit around waiting for something to happen. Instead, they spend much of their time traveling through and exploring the world around them. These actions play a decisive role in determining the kinds of stimuli they are repeatedly exposed to, as well as the frequency of exposure. In turn, these factors affect what the individuals learn about, what they remember, and how long the memories last.

Much of what is known about the learning and memory processes that occur when organisms inspect their surroundings comes from studies of object recognition and spatial navigation. Exposure to objects and places often does not initially lead to obvious changes in behavior; latent learning is the norm. Appropriately designed tests can, however, reveal a variety of short- and long-term effects of repeated exposures.

Recognizing Familiar Objects

Earlier, we noted that when a novel stimulus appears after repeated presentations of a familiar stimulus, babies show a strong orienting response to the new stimulus (because habituation is stimulus specific). A similar phenomenon can be seen in exploratory behavior. In the [novel object recognition task](#), for example, people, monkeys, or rodents are first allowed to freely

explore the room or box where the testing will be done, so that they become acclimated to the *context* of the experiment. Next, two identical objects or images are briefly presented within the experimental setting ([Figure 3.4A](#)). After a variable delay, one of these stimuli is presented again, this time paired with a new object or image ([Figure 3.4B](#)). Generally, individuals will spend about twice as much time examining the novel object as they do inspecting the familiar one. From this difference, researchers can infer that the individuals recognize the previously experienced object as one they have investigated before—that is, the repeated stimulus is perceived as familiar.

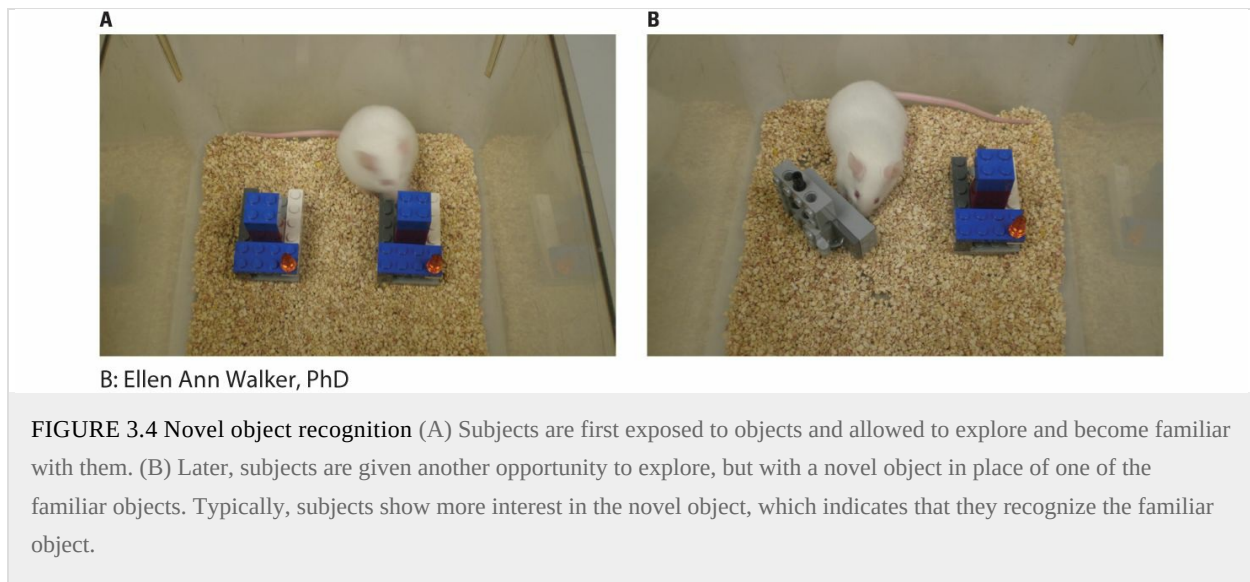


FIGURE 3.4 Novel object recognition (A) Subjects are first exposed to objects and allowed to explore and become familiar with them. (B) Later, subjects are given another opportunity to explore, but with a novel object in place of one of the familiar objects. Typically, subjects show more interest in the novel object, which indicates that they recognize the familiar object.

Description

The first photo shows a rat and two identical blocks inside in an experimental box. The rat is familiarizing with one of the blocks placed inside the box. The second photo shows the same rat familiarizing with a bigger box replaced inside the box.

novel object recognition task

A task in which an organism's detection of and response to unfamiliar objects during exploratory behavior are used to measure its memories of past experiences with those objects.

Not all animals are so eager to investigate novel objects, however. Some actively avoid them, a phenomenon known as *neophobia*. For instance, when dolphin trainers want to teach a dolphin to use an object, they often spend several sessions rewarding the dolphin for simply not bolting when the new object is brought near them (many dolphins are neophobic). In this case, the dolphin's fear response provides evidence that an object is not familiar.

Psychologists have used variants of the novel object recognition task to determine what kinds of information individuals remember about objects and places they have inspected in the past, such as an object's position and observable properties, and the circumstances under which it was observed. This task is also useful for investigating the resilience of memories formed through observations or exploration. The perception of *familiarity* is a fundamental component of memory. William James described memory in general as “*the knowledge of an event, or fact, of which meantime we have not been thinking, with the additional consciousness that we have thought or experienced it before*” [italicized as in [James, 1890, p. 648](#)]. **Familiarity** can be defined as the perception of similarity that occurs when an event is repeated. It is, in James's words, a “sense of sameness.” (By extension, *familiarization*, in the title of this chapter, refers to the acquisition of familiarity through repetition.)

familiarity

The perception of similarity that occurs when an event is repeated.

Although it is difficult to know what this feeling of sameness is like for a rat or monkey, we can conclude from tasks such as the novel object recognition task that rats and monkeys discriminate different levels of familiarity, just as they distinguish different frequencies of tones or wavelengths of light (abilities discussed in more detail in [Chapter 6](#) on generalization, discrimination learning, and concept formation). In the past decade, memory researchers have intensively investigated how individuals judge familiarity, how this capacity relates to other memory abilities, and what brain mechanisms contribute to familiarity judgments ([Eichenbaum, Yonelinas, & Ranganath, 2007](#)). Consequently, novel object recognition, which scientists once used mainly to distinguish habituation from fatigue, has transformed into a cornerstone of modern memory research.

Priming

Prior exposure to a stimulus can lead to a sense of familiarity the next time that stimulus is observed. Even when it does not lead to a conscious sense of familiarity, the prior exposure can still affect the individual's response to the repeated stimulus (or to related stimuli); this unconscious effect is called **priming**.

priming

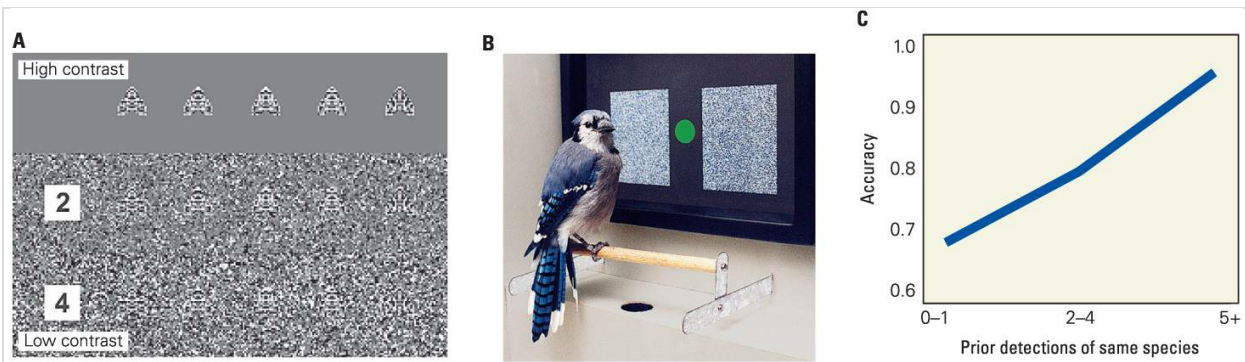
A phenomenon in which prior exposure to a stimulus can improve the ability to recognize that stimulus later.

Priming in humans is often studied using a [word-stem completion task](#), in which a person is given a list of word stems (MOT_____, SUP_____, and so on) and asked to fill in the blank with the first word that comes to mind. People generally fill in the blanks to form common English words (MOTEL or MOTOR, SUPPOSE or SUPPER). But if the people were previously exposed to a list of words containing those stems (MOTH, SUPREME, and so on), then they are much more likely to fill in the blanks to form words that were present in that list, even if they do not consciously remember having previously seen the words on the list ([Graf, Squire, & Mandler, 1984](#)). When they complete the word stem with a previously experienced word, they do not recognize the word as one they recently saw, and yet the prior experience clearly affects their word choice. Such effects are consistent with the findings from sexual habituation studies, described in “[Learning and Memory in Everyday Life](#)”, that repeated exposures can affect a person’s behavior and perception even if the person is not aware this is happening.

word-stem completion task

A task in which participants are asked to fill in the blanks in a list of word stems (e.g., MOT_____) to produce the first word that comes to mind; in a priming experiment, participants are more likely to produce a particular word (e.g., MOTEL) if they have been exposed to that word previously.

Nonhuman animals show priming, too. For example, blue jays like to eat moths, and moths have evolved coloration patterns that help them blend into the background where they alight. Therefore, blue jays have to be very good at detecting subtle differences in visual patterns that distinguish a tasty meal from a patch of tree bark. Researchers studied this detection ability by training blue jays to look at pictures on a screen ([Figure 3.5A](#)) and to peck at the screen to signal “there’s a moth here” or at a key to signal “no moth” ([Figure 3.5B](#)). The birds did very well, but they were quicker and more accurate at detecting a particular species of moth if they had recently detected other members of that species, as shown in [Figure 3.5C](#) ([Bond & Kamil, 1999](#)). In other words, recent observations of one kind of moth *primed* the jays’ abilities to recognize similar moths later.



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A: Illustrations by Alan B. Bond, © 1999; B: Alan B. Bond University of Nebraska Lincoln

FIGURE 3.5 Priming in blue jays (A) Virtual moths on a gray background are more easily detectable than the same images of moths on speckled backgrounds. Higher numbers at left indicate more cryptic backgrounds. (B) Blue jays learn to peck on screens when they detect a virtual moth and to peck on a green key when they detect no moths. (C) The blue jays are better able to detect a moth if it is similar to a recently detected one, suggesting that prior exposure facilitates detection. In other words, priming has occurred.

Description

The first illustration labeled A shows virtual moths in two backgrounds, high contrast and low contrast. The area labeled High Contrast has a line of virtual moths. The area labeled Low contrast shows a speckled screen, with the numbers 2 and 4.

The second illustration labeled B shows a blue jay, sitting on a rod, facing two screens. Both the screens have speckles.

The third illustration shows a graph plotting Accuracy versus Prior detections of same species. The horizontal axis has Prior detections of same species. The vertical axis has Accuracy. The data in the graph are as follows. The line starts at 0.68, and it gradually increases to 0.93.

As noted above, priming can occur even in the absence of any feelings of familiarity or recognition that a stimulus was previously experienced. In addition, priming effects may persist much longer than recognition of past encounters ([Tulving, Schacter, & Stark, 1982](#)). These findings led some researchers to propose that priming and recognition involve independent processes ([Tulving & Schachter, 1990](#)). More recent behavioral studies indicate, however, that a single memory process might be able to account for both priming- and recognition-related effects of repetition ([Berry, Shanks, Speekenbrink, & Henson, 2011](#)).

Questions about the number of different processes that are required to explain different phenomena have loomed large in experimental and theoretical studies of learning and memory. Even in “simple” cases where you repeatedly experience an object or a word, a currently

unknown number of learning and memory processes may be engaged. These so-called simple scenarios are further complicated by the fact that repeatedly experiencing a stimulus can change how you perceive the stimulus, a phenomenon known as *perceptual learning*.

Perceptual Learning

Learning in which repeated experiences with a set of stimuli make those stimuli easier to distinguish is called **perceptual learning**. Consider the making of medical diagnoses. All rashes may look alike to an inexperienced medical student, but an experienced dermatologist can glance at a rash and tell immediately, and with high accuracy, whether a patient has contact dermatitis, ringworm, or some other condition. You too may have perceptual expertise in making certain kinds of precise sensory distinctions. Perhaps you can easily distinguish Coke from Pepsi or McDonald's french fries from those of any other fast-food restaurant. Maybe for you the sound of a Fender electric guitar is very different from the sound of a Gibson. Whatever your specialty, you likely developed your capacities to detect subtle differences through repeated exposures to those stimuli. This is the essence of perceptual learning.

perceptual learning

Learning in which experience with a set of stimuli makes it easier to distinguish those stimuli.



Keith Goldstein/ Photodisc/
Getty Images

Through extensive experience, gem assessors become attuned to subtle visual differences in stones. In what ways could mere exposure learning assist in this endeavor, and in what ways might it be insufficient?

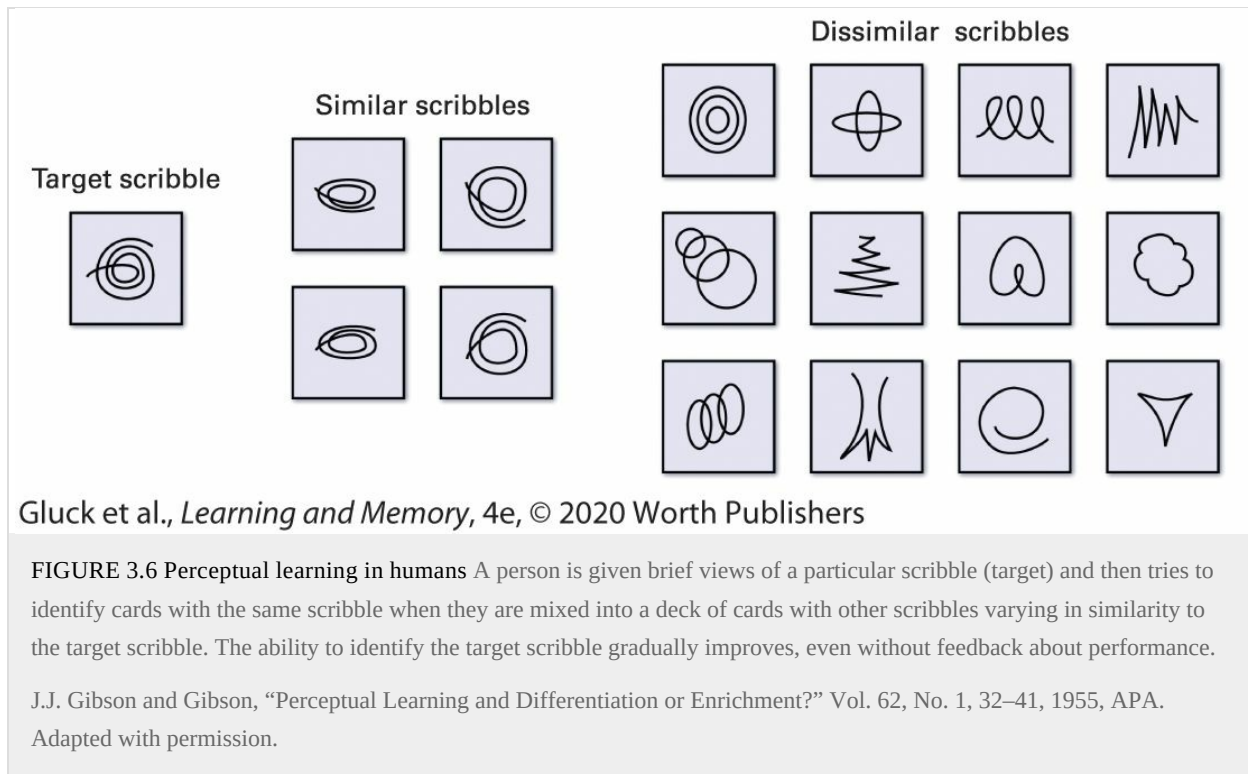
Dermatologists gain their expertise at diagnosing rashes not by accident but through extensive, deliberate practice. This type of perceptual learning is discussed more extensively in [Chapter 8](#). Sometimes, however, perceptual learning happens through mere exposure to stimuli, without any conscious effort by the individual—a situation similar to what occurs during habituation, sensitization, and priming. For example, Eleanor Gibson and colleagues exposed one group of rats to large triangular and circular shapes mounted on the walls of their home cages for about a month ([Gibson & Walk, 1956](#)). The researchers then trained this group and a control group of rats to approach one of the shapes but not the other. Rats familiar with the shapes learned to discriminate between them faster than rats that had never seen the shapes before. During the initial, exposure phase, nothing was done to teach the rats in the experimental group about the shapes, and the shapes were irrelevant to the rats' daily activities. Nevertheless, experience viewing the shapes facilitated later learning about them. This learning process is a type of latent learning (which we discussed at the chapter's beginning) because before the rats begin training with the shapes, there is no behavioral evidence that they have learned anything about the shapes that they saw in their home cages. This initial learning became evident only when the rats were explicitly trained to respond in different ways to the two different shapes. Perceptual learning that happens without explicit training is sometimes called [statistical learning](#) because the percepts that individuals learn the most about are those that are experienced most frequently and consistently ([Aslin, 2017](#)).

statistical learning

Learning driven by repeated exposures to perceptual events that increases the familiarity and distinctiveness of those events.

People, too, show perceptual learning after repeated exposure in the laboratory. In one study, volunteer participants learned to discriminate between complex line drawings—the scribbles shown in [Figure 3.6](#). First, each participant was asked to look briefly at a card containing a target scribble. Then the participant was shown a series of individual cards that each contained a scribble and was told that some of the cards contained the same scribble shown on the first card. As each card was presented, the participant was asked to tell the experimenter whether it matched the target scribble. The experimenter gave no feedback—no indication of whether a participant's answer was correct or not—but at the end of the process, the same steps (beginning

with the scrutiny of the same target card) were repeated all over again, after which they were run through a third time.



Description

The first illustration, toward the left, shows a card with a coil-shaped pattern. It is labeled, Target scribble.

The second illustration, in the middle, shows four cards with multiple shapes. All the four cards have a similar coil-pattern. It is labeled Similar scribbles.

The third illustration, in the right, shows eight cards with multiple shapes. Each card has a distinct shape. It is labeled Dissimilar scribbles.

Initially, participants were pretty accurate at identifying repetitions of the target scribble, as well as at rejecting novel scribbles that were very unlike the target (“dissimilar scribbles”). Early in the experiment, however, participants would also incorrectly “recognize” many scribbles that were similar to the target scribble. Eventually, with more and more exposure to scribbles, the participants began to distinguish the target scribble from very similar but novel stimuli ([Gibson & Gibson, 1955](#)). This is an example of perceptual learning through mere exposure to scribbles. (Your ability to distinguish the faces of certain groups of people better than the faces of other

groups may have developed similarly, perhaps leading to problems of unconscious bias; see “[Learning and Memory in Everyday Life](#)”.)

LEARNING AND MEMORY IN EVERYDAY LIFE

Unconscious Racial Bias

People typically judge the faces of individuals from another racial group to be more similar to one another than are the faces of unfamiliar individuals from their own race ([Malpass & Kravitz, 1969](#)). This bias is called the “other-race effect.” Current psychological theories suggest that the other-race effect occurs when people have more experience with faces from certain racial groups than from others. Consistent with this idea, non-Caucasian children who are adopted by Caucasian families show better discrimination of Caucasian faces than of faces from members of their own racial group ([Meissner & Brigham, 2001](#)).

You may like to think that you treat all strangers equally, without taking into account appearance or speech patterns. But laboratory experiments suggest that this is unlikely. Despite people’s stated ideals, they also often show racial biases in situations other than the distinguishing of faces. For example, people often show evidence of unconscious negative associations when answering questions about racial groups other than their own ([Greenwald, McGhee, & Schwartz, 1998](#)). These biases are not predictable from their self-reports. You can try some of these unconscious racial bias experiments yourself online at <https://implicit.harvard.edu/implicit/> if you are curious to see how you fare.

People’s insensitivity to facial differences among members of other races can actually contribute to unconscious racial biases ([Lebrecht, Pierce, Tarr, & Tanaka, 2009](#)). The explanation offered is that the less able a person is to reliably differentiate individuals of a certain racial classification, the more likely the person is to unconsciously apply generic racial stereotypes to all individuals of that group.

If the ability to distinguish faces is a consequence of the frequency with which different types of faces are encountered, then the other-race effect is essentially a side effect of visual perceptual learning. In that case, you might expect that if you trained individuals to distinguish faces of racial groups they are not familiar with, you might be able to reduce the other-race effect. Furthermore, if the other-race effect contributes to unconscious racial biases, such training might reduce those biases as well. This prediction has been confirmed in laboratory experiments. Caucasian participants who were extensively trained to distinguish between faces of African American individuals showed both a reduction in the other-race effect and a reduction in unconscious racial biases ([Lebrecht et al., 2009](#)). Apparently, learning to recognize individuals of other racial groups can reduce overgeneralization of racial stereotypes, even when that learning involves no social interactions that might counteract the stereotypes. (See [Chapter 6](#) for further discussion of the origins of differential processing of race-related stimuli.)

Perceptual learning is similar to priming in that it leads to more effective processing on subsequent encounters with the stimuli. In addition, perceptual learning can happen even if the learner is not aware that her sensitivities to perceptual differences are increasing. One way in which perceptual learning differs from priming is that the resulting increase in the ability to tell similar stimuli apart is long lasting (discussed further in [Chapter 6](#)). Priming is more often associated with short-term changes in the detection and recognition of recently experienced

stimuli.

Habituation, sensitization, and perceptual learning might seem at first glance to be very different kinds of learning. However, current theories suggest that these seemingly different phenomena depend on similar (or identical) learning mechanisms. For example, the *dual process theory* of habituation, introduced earlier in the chapter, explains perceptual learning as resulting from a combination of habituation and sensitization processes ([Hall, 2009](#)). Consider again the experiment on mere exposure learning, in which rats lived in a cage with triangles and circles on the walls ([Gibson & Walk, 1956](#)). The triangles and circles shared some features: both shapes were constructed of the same material, both were located on the walls (but never, for example, on the floor of the cage), and so on. They also differed in some features: for instance, the triangles had straight sides, and the circles had round sides. According to dual process theory, when the rat viewed a triangle on the wall, its orienting responses and responses to novelty (like neophobia) habituated to all of the features it experienced. Similarly, its responses also habituated to all visual aspects of the circle on the wall.

Notice that the shared features of the shapes were experienced every time the rat viewed either shape but that distinguishing features were experienced only when the rat viewed one shape or the other. Since the shared features were repeatedly experienced twice as often, the effects of viewing those shared features would have been greater: there would be twice as much habituation of responses to the shared features as to the unshared features. The end result would be that distinctive features (such as straight versus round edges) would be more likely to provoke orienting responses than would shared features. In other words, for rats that lived with the shapes, the features that distinguished the two shapes became more noticeable than the shared features. Thus, rats that lived with the shapes would be expected to learn to distinguish the shapes faster than rats that had never seen the shapes before because the experienced rats should pay less attention to the shared features of the shapes.

TEST YOUR KNOWLEDGE

Perceptual Learning Versus Habituation

Both habituation and perceptual learning can result from repeated exposures to stimuli. Although the experiences that lead to these phenomena can be similar, the kinds of responses that provide evidence of these two forms of learning are notably different. For each of the following photographs, identify what kind or kinds of learning might have contributed to the scene depicted. ([Answers appear at the end of the chapter.](#))



Top left: Sean Murphy/The Image Bank/Getty Images; right: Sheer Photo, Inc./Photodisc/Getty Images; bottom: Ian Shaw/The Image Bank/Getty Images

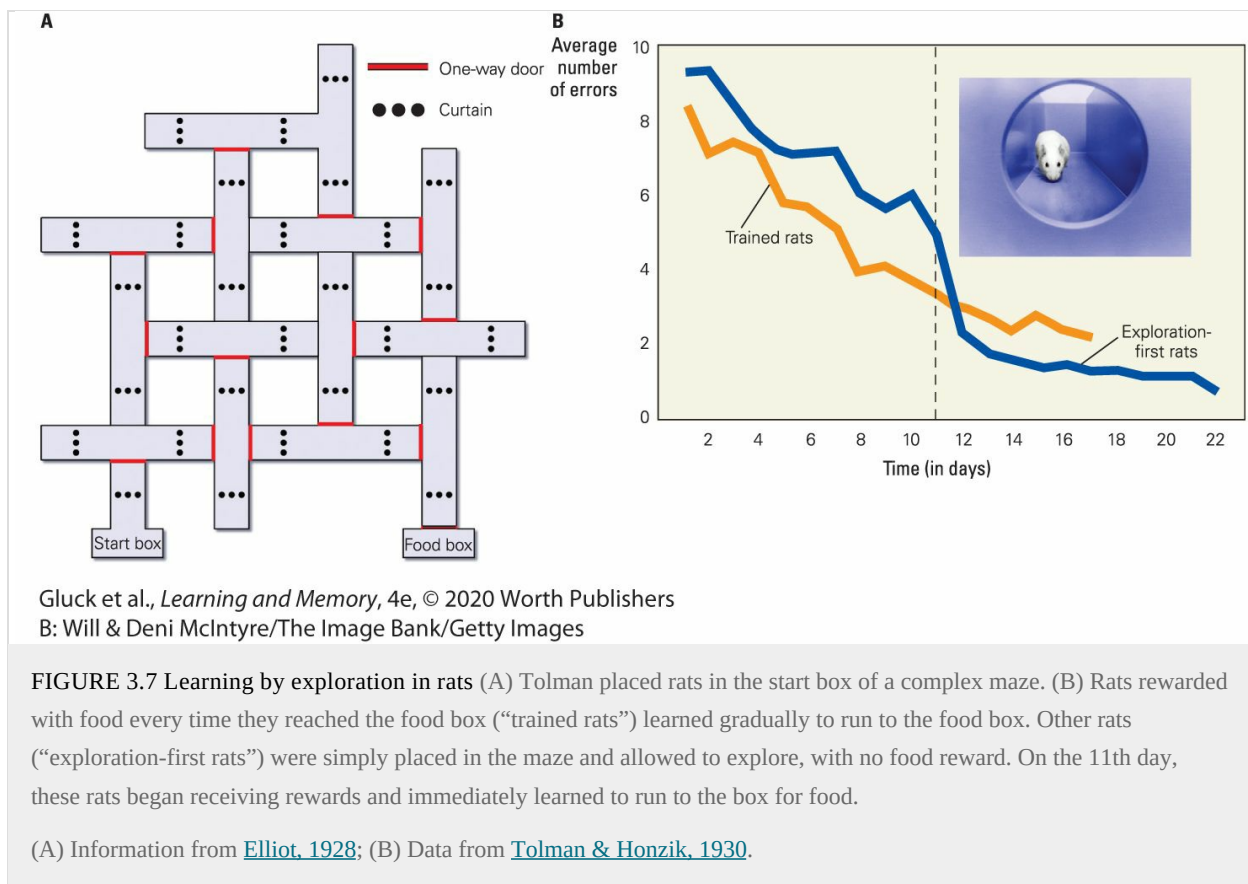
Description

The first photo shows a close-up view of the mouth of a dog, held open by two people. The second photo shows a man holding a glass of wine. The third photo shows the close-up of the legs of a group of people walking on the street. A beggar is also shown sitting down on the street.

Spatial Learning

When animals explore, they learn more than just what objects look, smell, and sound like. They

also learn how to get from one place to another and what to expect when visiting particular places. **Spatial learning**—the acquisition of information about one’s surroundings—can be accomplished in different ways. In one of the earliest laboratory studies of exploratory spatial learning, Edward Tolman, working with C. H. Honzik, placed rats in a complex maze ([Figure 3.7A](#)) and trained them to make their way to a particular location in it—the food box—to be rewarded with a bit of food ([Tolman & Honzik, 1930](#)). These trained rats learned to run to the food box with fewer and fewer errors (wrong turns) as the days went by ([Figure 3.7B](#)). Rats in a second group were simply placed in the maze for the first 10 days of the study and allowed to explore. If they happened to enter the food box, they received no food but were removed from the maze. On the 11th day, these rats started getting food every time they entered the food box. As [Figure 3.7B](#) shows, these exploration-first rats also learned to run to the food box to get their food—and they learned this task so well that their performance quickly surpassed that of the rats who had been training on the task all along!



Description

The first illustration labeled A shows the design of a complicated maze. It has a start box and a Food box. The legends

of the maze are as follows. A line represents the one-way door, and the three dots represents the curtain in the maze.

The second illustration labeled B shows a line graph. The horizontal axis shows Time in days. The vertical axis shows the Average number of errors. A dotted line is drawn from 11, on the horizontal axis.

The data of the graph are as follows. Two lines are plotted on the graph. The first line is labeled Trained rats. It starts from (1.8, 8) and gradually decreases to (17, 2). A second line labeled Exploration-first rats starts from (1.8, 9), decreases to (10, 6), and ends at (22, 1). Both the line crosses each other at a point (12, 3). All data are approximate.

A photo at the top-right corner of the illustration shows a rat walking through a maze.

spatial learning

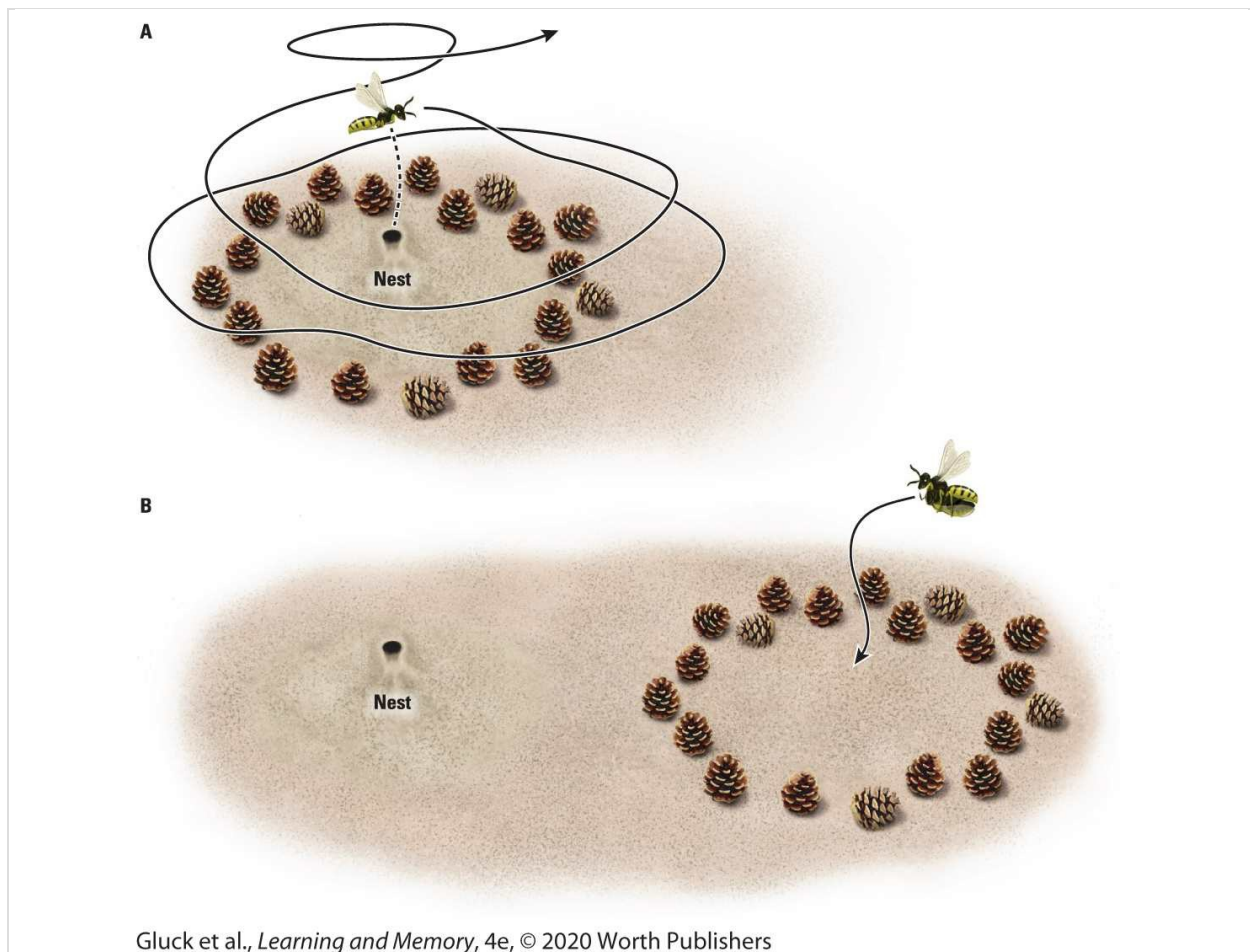
The acquisition of information about one's surroundings.

Tolman and Honzik concluded that both groups of rats had learned about the location of the food box. One group had learned how to get to the food box through explicit training, and the other group learned the spatial layout of the maze simply by exploring it. This is yet another example of latent learning because, until food was placed in the food box, there was little evidence that the explorer rats knew how to get there quickly.

What were the rats learning? Perhaps they were learning a sequence of turns that they could perform to get to certain spots: turn right from the start box, then left, and so on. Such learning does occur, but it isn't enough to account for everything the rats did in the maze because a rat could be placed in a new start position and still find its way to the goal. Rats also appear to use visual cues to determine their location in such mazes. For example, a rat in a laboratory maze may use the sight of a window or a wall decoration visible over the side of the maze to orient itself. As long as these cues are in sight, the rat may learn to navigate from any starting point in the maze; if the cues are moved around, however, the rat may get temporarily confused (we describe such an experiment later in the chapter). In fact, rats can learn many things while exploring a maze; as a result, spatial tasks have played a major role in studies of many different kinds of learning and memory, including studies of operant conditioning ([Chapter 5](#)), memories for events ([Chapter 7](#)), skill memories ([Chapter 8](#)), and working memory ([Chapter 9](#)). Exploration is important in these tasks because a rat that refuses to explore a maze will learn very little about its spatial organization.

Many kinds of animals in the wild also navigate based on visual cues they have learned to recognize through exploration. In a classic study, Niko Tinbergen examined wasps' ability to

locate their home nest. Before leaving their hives or burrows to look for food, certain species of wasps and bees engage in orientation flights during which they circle their home base. In one experiment, Tinbergen and William Kruyt laid a circle of pinecones around a wasp burrow while the wasp was inside (Tinbergen & Kruyt, 1972). The experimenters left the pinecone circle intact for several orientation flights—long enough for the wasp to learn to recognize these landmarks (Figure 3.8A). Then, while the wasp was away on a foraging trip, the experimenters moved the circle of pinecones away from the burrow (Figure 3.8B). When the wasp returned, it repeatedly searched for its burrow within the ring of pinecones.



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FIGURE 3.8 Use of landmarks by wasps (A) Tinbergen and Kruyt placed pinecones around a wasp's burrow (an underground nest) to provide visual information about the burrow's location. When leaving home, wasps take orientation flights, during which they seem to note local landmarks (such as the pinecones) that will help them return. (B) When the circle of pinecones was moved to flat ground near the nest, the returning wasp searched for the burrow inside the circle of pinecones.

Information from [Tinbergen, 1951](#).

Description

The illustration labeled A shows a wasp flying over its nest among the pinecones. The illustration labeled B shows a bee searching for its nest in the middle of the pinecones. The pinecones are moved away from the nest.

Tinbergen and Kruyt concluded that when wasps leave home to forage, they use the orientation flight to collect visual information about landmarks that will later help them locate the burrow. If these landmarks are repositioned while the wasp is away, the wasp will search for the burrow based on the landmarks, revealing that it has learned about the spatial relationship between the burrow and surrounding landmarks. Just like Tolman's rats, the wasps learn about the spatial properties of their environments through exploration.

As illustrated in [Figure 3.8](#), wasps often make several passes over the nest before departing. After multiple trips, a wasp will have repeatedly experienced the visual cues surrounding its nest. Such repeated events could lead to habituation, sensitization, perceptual learning, or other forms of learning ([Table 3.1](#)). In this sort of naturalistic experiment, there is no way to know exactly what a wasp is learning from its experiences. After exploring the neurological bases of habituation and sensitization, [Section 3.2](#) describes other, more controlled studies that are helping researchers get a better handle on what individuals learn from repeated exposures to stimuli during exploration.

TABLE 3.1 Learning and memory processes that can be engaged by repetition

Process	Example
Habituation	After repeated trips on airplanes, you no longer notice when babies start crying.
Sensitization	After repeated trips on airplanes, you start pulling out your hair when babies start crying.
Familiarization	Passing the same person on your walk every day leads to a feeling of familiarity when you see her.
Priming	You see a commercial for junk food and later crave junk food as a result, without recalling the commercial.
Perceptual learning	You work for a year at a coffee shop and learn to identify brands of coffee from their odors.
Spatial learning	You work at a coffee shop and become able to tell customers where to find specific brands of coffee.

Interim Summary

- Habituation is a process in which repeated exposure to a stimulus leads to a decrease in the

strength or frequency of the responses evoked by that stimulus.

- In sensitization, exposure to an arousing stimulus causes a heightened response to stimuli that follow.
- Dual process theory proposes that behavioral changes caused by repeated exposures to a stimulus reflect the combined effects of habituation and sensitization.
- In novel object recognition tasks, an organism typically responds more to a novel stimulus than to a stimulus it was previously exposed to, providing evidence of familiarity.
- Priming is a phenomenon in which exposure to a stimulus, even without a conscious memory of that exposure, affects the organism's response to the stimulus later.
- Perceptual learning occurs when repeated experiences with a set of stimuli improve the organism's ability to distinguish those stimuli.
- Spatial learning often involves latent learning about features of the environment (including encountered objects) through exploration.

3.2 Brain Substrates

The preceding discussion considers several ways in which repeated exposures to stimuli can lead to learning. In some cases, gradual changes in behavior reveal this learning, but in others the learning is latent until the right test is given. Even in the “simple” case of habituation, learning seems to involve multiple parallel processes that can sometimes operate without any associated changes in behavior. As a consequence, it is extremely difficult to understand how and what organisms learn from repeated events on the basis of behavioral evidence alone. Fortunately, the ability to study changes in brain function and structure as individuals learn about repeated events has provided clues to the nature of these phenomena, as the following discussions illustrate.

Any dog or cat owner knows that dogs and cats are natural antagonists. Some of the earliest brain studies on habituation, using dogs and cats as subjects, seemed to bear out the view that these animals are antithetical even at the level of brain anatomy. Ivan Pavlov, for example, found that when a dog’s cortex was removed, the dog no longer showed habituation to auditory stimuli: the dog would instead continue to show orienting responses to the sounds, even after many exposures ([Pavlov, 1927](#)). Such findings led researchers to suggest that the cortex is critical for habituation and that it actively suppresses reflexive orienting responses to stimuli perceived as familiar ([Sokolov, 1963](#)). Behavioral data from cats, however, seemed to contradict these findings completely. Cats that had had their brain disconnected from their spinal cord, called *spinal cats*, still showed habituation to tactile stimulation ([Thompson & Spencer, 1966](#)). This seemed to prove that the spinal cord by itself contains all the neural machinery necessary for habituation; the cortex—and indeed the rest of the brain—isn’t needed.

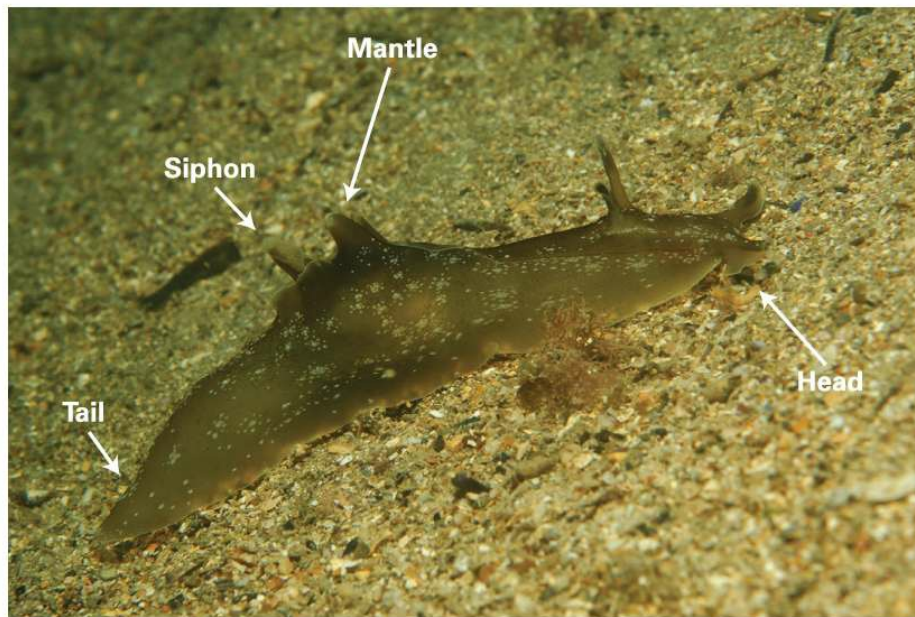
Can these findings about cats and dogs be reconciled? Easily! For one thing, the animals in these early studies were learning about different kinds of stimuli. Whether cortical processing is involved in habituation likely depends on the kinds of stimuli that are being repeated, where they are normally processed, and where memories of the stimuli are formed. One way to avoid these complications is to study habituation not in mammals but in smaller-brained animals, such as the laboratory animal that is a favorite of neuroscientists—the sea hare.

An Invertebrate Model System

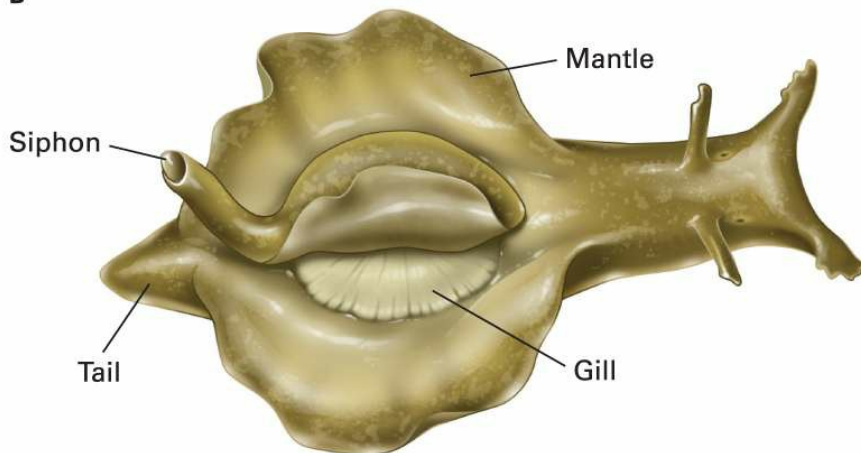
Much work on the neural substrates of habituation has been conducted on a group of marine

invertebrates called *Aplysia* (pronounced “a please yah”), the sea hares (or sea slugs), such as the species *Aplysia californica* shown in [Figure 3.9](#). Like many other marine animals, *Aplysia* breathe through gills, which extend upward from between the two wings of the mantle, the animal’s outer covering. A structure called the *siphon* works like a tube to blow aerated water over the gills to assist respiration. The gills are delicate and easily damaged. When danger threatens, the *Aplysia* tends to retract them under the safety of the mantle—a reaction known as the *gill-withdrawal reflex*.

A



B



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Borut Furlan/WaterFrame/Getty Images

FIGURE 3.9 *Aplysia californica*, the sea hare (A) This marine invertebrate, a shell-less mollusk, has a relatively simple nervous system that is useful for studying the neural bases of learning. (B) Schematic of how *Aplysia* are prepared for experiments. If the siphon is touched lightly, both siphon and gill are protectively withdrawn (the gill-withdrawal reflex). With repeated light touches, the gill-withdrawal reflex habituates. In this photo, the gill is underneath the mantle.

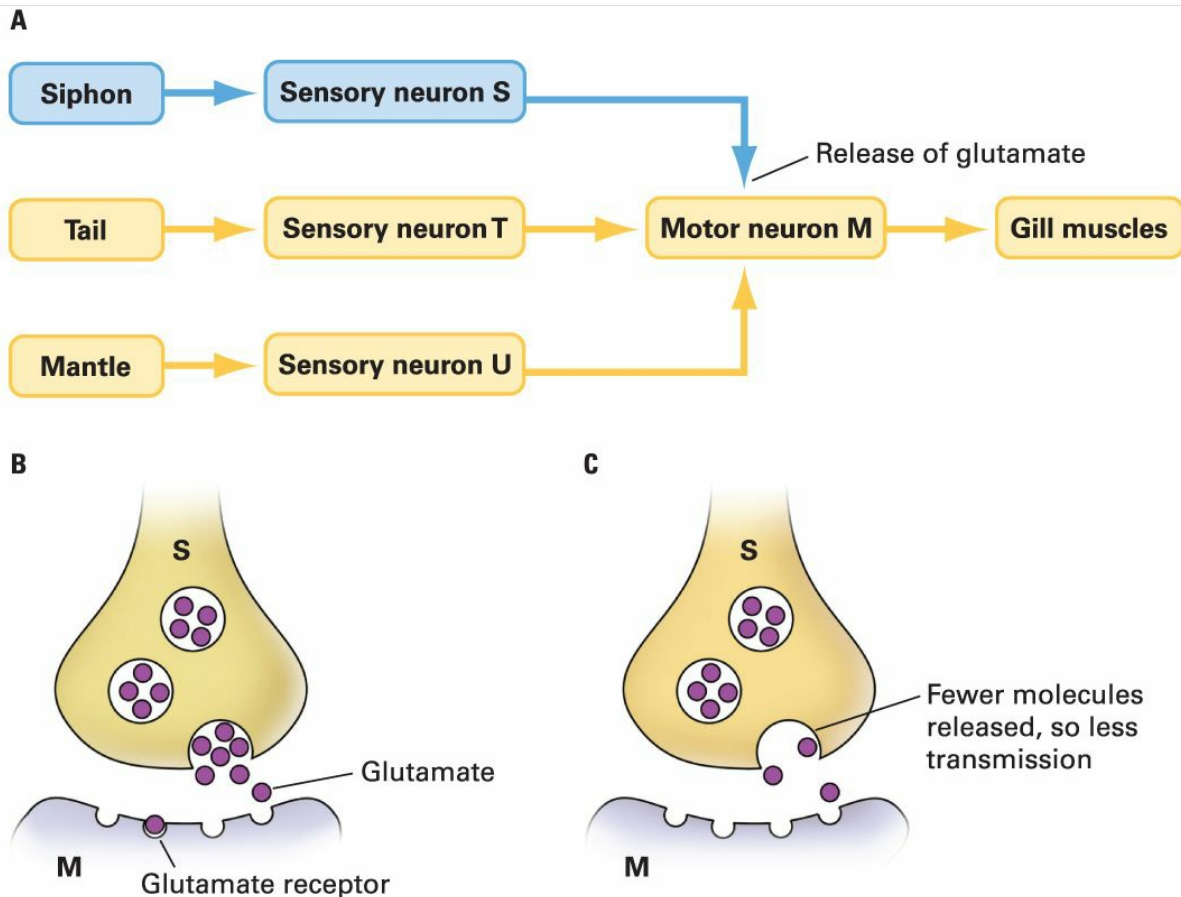
Information from [Squire & Kandel, 2000](#).

Description

The first photo shows a shell-less mollusk, with its body parts labeled. The parts labeled are Head, Mantle, Siphon, and Tail. The second photo shows the labeled anatomy of the upside down view of a mollusk. The parts labeled are Siphon, Tail, Gill, and Mantle.

One advantage of studying the learning process in *Aplysia* is that these slugs have a relatively simple nervous system—only about 20,000 neurons, compared with the tens of billions in a cat or human. Plus, some of the neurons are very big. A few are large enough to be seen with the naked eye. Best of all, the neural circuits in *Aplysia* seem to be “hardwired,” meaning that researchers can identify a particular neuron in one *Aplysia* (say, motor neuron L7G) and find the same neuron in the same place in another member of the species. This type of nervous system makes it much easier for a neuroscientist to understand how the brain changes with learning.

Neuroscientists have documented each of the neurons involved in *Aplysia*’s gill-withdrawal reflex. The siphon contains 24 sensory neurons that are directly connected to 6 motor neurons that control gill movements. [Figure 3.10A](#) shows a simplified scheme of this system of neurons, consisting of three sensory neurons, S, T, and U, and one motor neuron, M. When the siphon is touched, sensory neuron S fires, releasing a neurotransmitter, *glutamate*, into the synapse ([Figure 3.10B](#)). Molecules of glutamate diffuse across the synapse to activate receptors in motor neuron M. If enough receptors are activated, neuron M fires, causing the muscles to retract the gill for a few seconds.



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FIGURE 3.10 Neural circuits in *Aplysia* gill-withdrawal reflex (A) Sensory neurons S, T, and U respond to a touch on the siphon, tail, and upper mantle, respectively. These neurons converge on motor neurons such as M, which contract the gill muscles. (B) When sensory neuron S fires, it releases the neurotransmitter glutamate into the synapse between S and M. The glutamate molecules (shown in purple) may dock at receptors on neuron M. If enough receptors are activated, neuron M will fire, retracting the gill. (C) If neuron S is activated repeatedly, it gradually releases less glutamate each time, decreasing the response of M. This synaptic depression underlies habituation of the gill-withdrawal response in *Aplysia*.

Description

The first illustration A shows a flow diagram of the neural circuits in *Aplysia* gill-withdrawal reflex. The first layer of the flow begins with a text box that reads, Siphon. It leads to Sensory neuron S, which further leads to Motor neuron M. It finally leads to a text box that reads, Gill muscles. Release of glutamate occurs when Sensory neuron moves on to Motor neuron.

The middle layer of the flow begins with a text box that reads, Tail. It leads to Sensory neuron T, which further leads to Motor neuron M. Finally, it leads to Gill muscles. The last layer of the flow begins with a text box that reads, Mantle which leads to Sensory neuron U. This leads to Motor neuron M, which finally leads to Gill muscles.

The second illustration B shows the sensory neuron S firing neurotransmitters, Glutamate into the synapse between S and M. A label marked Glutamate docks at the base receptors on the neuron M.

The third illustration C shows the sensory neuron S firing neurotransmitters, into the synapse between S and M. A label marked, Fewer molecules released, so less transmission, points to a dock at the base receptors on the neuron M.

As simple as *Aplysia* are, however, they are still capable of adapting their behavior in response to experience. *Aplysia* show habituation, sensitization, and several other forms of learning, just as rats and humans do. But in *Aplysia*, scientists can actually watch the nervous system in action as these learning processes occur. Nobel Prize winner Eric Kandel pioneered the use of *Aplysia* reflexes as a way of exploring how repeated experiences change the neural circuits that generate those reflexes, especially in situations where animals become habituated or sensitized to tactile stimuli.

Habituation in *Aplysia*

An initial, light touch on an *Aplysia*'s siphon activates the gill-withdrawal response, but if the touch is repeated, the gill-withdrawal reflex gradually becomes weaker, or habituates. The amount of habituation is proportional to the intensity of the stimulus and the repetition rate. If a sufficiently light touch is delivered every minute, the withdrawal response habituates after 10 or 12 touches, and the habituation can last for 10 to 15 minutes ([Pinsker, Kupfermann, Castellucci, & Kandel, 1970](#)).

In *Aplysia* we can see exactly what is causing this habituation. Refer back to the schematic diagram in [Figure 3.10A](#). Recall that touching the siphon excites sensory neuron S, which releases the neurotransmitter glutamate, which in turn excites motor neuron M, which drives the withdrawal response ([Figure 3.10B](#)). With repeated stimulation, however, neuron S releases less glutamate ([Figure 3.10C](#)), decreasing the chance that neuron M will be excited enough to fire ([Castellucci & Kandel, 1974](#)). The reduction in glutamate release is evident even after a single touch and lasts for up to 10 minutes. This decrease in neurotransmitter release is associated with a decrease in the number of glutamate-containing vesicles positioned at release sites. Thus, in *Aplysia*, habituation can be explained as a form of **synaptic depression**, a reduction in synaptic transmission. This is exactly the sort of weakening of connections that is proposed by the dual process theory of habituation (see [Figure 3.3B](#)) and that is thought to contribute to the *long-term depression* of neural connections described in [Chapter 2](#).

synaptic depression

A reduction in synaptic transmission; a possible neural mechanism underlying habituation.

An important feature of habituation in *Aplysia* is that it is **homosynaptic**, which means it involves only those synapses that were activated during the habituating event; changes in neuron S do not affect other sensory neurons, such as T or U in [Figure 3.10A](#). In other words, a light touch to the tail or upper mantle still elicits the defensive gill withdrawal, even though a touch to the siphon is ignored. The responsiveness of the motor neuron M is not changed. In this case, habituation in the short term affects only how much neurotransmitter neuron S releases.

homosynaptic

Occurring in one synapse without affecting nearby synapses.

Habituation in *Aplysia* can often last much longer than 10 minutes, especially when exposures are spaced over several days ([Cohen, Kaplan, Kandel, & Hawkins, 1997](#)). How are the animals storing information about past exposures for such a long time? When an *Aplysia* is repeatedly exposed to the same stimulus over several days, the actual number of connections between the affected sensory neurons and motor neurons decreases. Specifically, the number of presynaptic terminals on the sensory neurons of animals that have been repeatedly exposed to the same stimulus is reduced. Synaptic transmission in *Aplysia* can thus be depressed not only by decreases in neurotransmitter release but also by the elimination of synapses. This suggests that repeated experiences can lead not only to the weakening of connections, as suggested by the dual process theory of habituation, but also to their elimination.

Do the mechanisms of habituation in *Aplysia* tell us anything about habituation in people? It is currently impossible to trace the entire neuronal circuit of habituation through the billions of neurons in a mammalian brain. However, neuroscientists have good reason to believe that the mechanisms of habituation documented in *Aplysia* occur in other species, too. In fact, repeated stimulation of sensory neurons in other species, including crayfish and cats, also causes a reduction in neurotransmitter release. This suggests that at least some of the biological mechanisms of habituation are comparable across species.

Sensitization in *Aplysia*

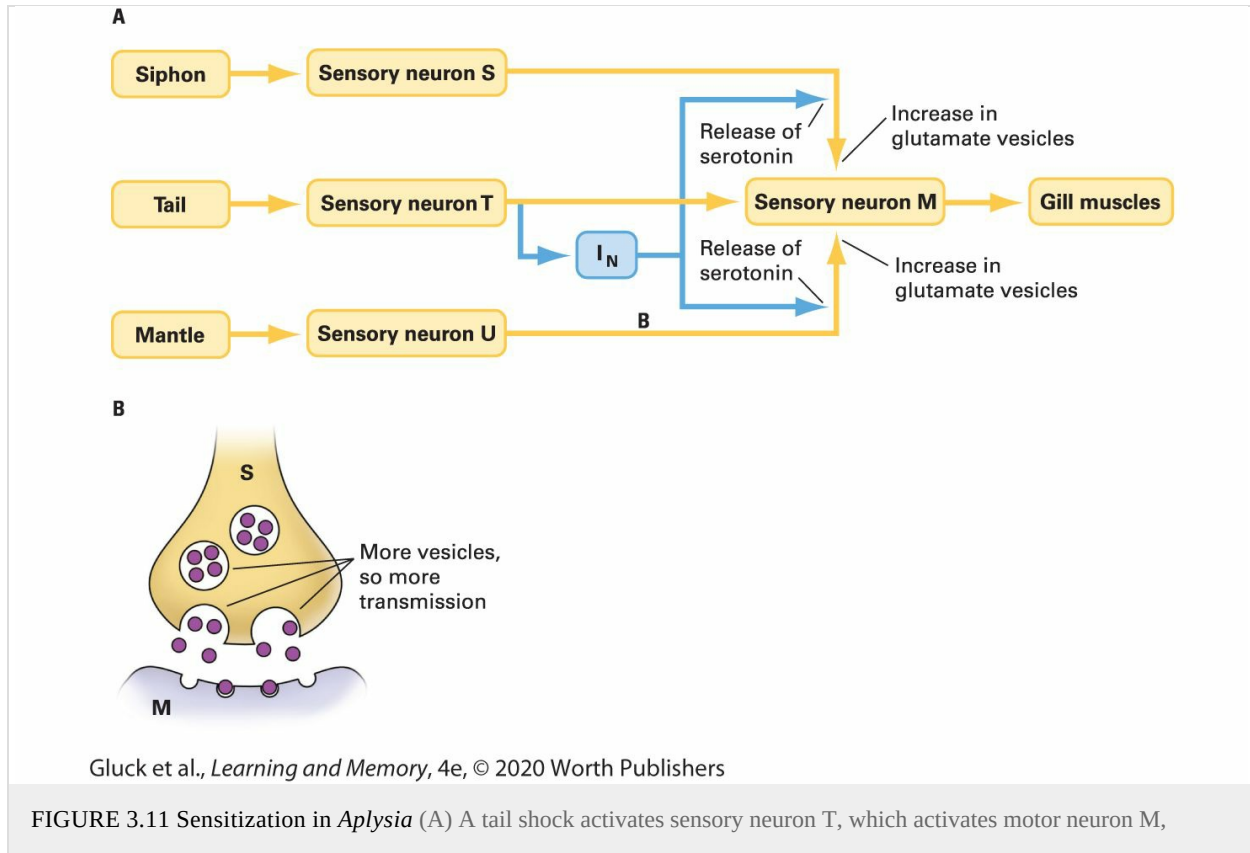
What about sensitization, which, in contrast to habituation, *increases* responses to stimuli?

Aplysia also provide a way to study the neural processes involved in this kind of learning.

Suppose that instead of applying a light touch to the siphon, the researcher applies a more unpleasant stimulus: a mild electric shock to the tail that causes a large, sustained gill-withdrawal

response. The aversive tail shock sensitizes subsequent responding, such that a weak touch to the siphon now produces a strengthened gill withdrawal.

To understand how this occurs, let's take the simplified circuit diagram from [Figure 3.10A](#) and add one more level of neural detail, as shown in [Figure 3.11A](#). The tail shock activates sensory neuron T, which activates motor neuron M, causing the gill-withdrawal response. But because of the arousing nature of shocks, neuron T also activates modulatory interneurons, such as I_N . An *interneuron*, as its name suggests, is a neuron that neither directly receives sensory inputs nor produces motor outputs but instead acts as an “intermediary,” carrying a message between two other neurons. A *modulatory interneuron* alters the strength of the message being transmitted. [Chapter 2](#) describes *neuromodulators*, which are neurotransmitters that can affect activity in entire brain areas rather than just at a single synapse. In *Aplysia*, interneuron I_N connects neuron T to both S and U, communicating with them by releasing a neuromodulator such as *serotonin*. Serotonin increases the number of glutamate vesicles available to release glutamate from neuron S each time it fires. In effect, the interneuron does not tell S whether to fire; rather, it tells S, “When you do fire, fire strongly.”



causing the motor response. T also activates interneuron I_N , which delivers a neuromodulator (such as serotonin) to the axons of neurons S and U. (B) Subsequent activation of neuron S will cause a larger release of neurotransmitter (glutamate), leading to greater activation of neuron M than was previously evoked by S.

Description

The first illustration A shows a flow diagram of the Sensitization in *Aplysia*. The first layer of the flow begins with a text box that reads, Siphon. It leads to Sensory neuron S, which further leads to Sensory neuron M. It finally leads to a text box that reads, Gill muscles. Increase in glutamate vesicles occurs when Sensory neuron moves on to Motor neuron. The middle layer of the flow begins with a text box that reads, Tail. It leads to Sensory neuron T. An arrow pointing from the text box, Sensory neuron T, reads, I_N subscript N. It further branches into two reading, Release of serotonin and Release of serotonin. The flow continues to a text box that reads Sensory neuron M. It further leads to, Gill muscles. The last layer of the flow begins with a text box that reads, Mantle, which leads to Sensory neuron U. It leads to Sensory neuron M, which finally, leads to Gill muscles. Increase in glutamate vesicles occurs when sensory neuron U moves on to Sensory neuron M.

The second illustration B shows the sensory neuron S firing neurotransmitters into the synapse between S and M. A label marked, More vesicles, so more transmission, docks at the base receptors on the neuron M.

Suppose the *Aplysia* now experiences a mild touch to the siphon. Before the tail shock, this mild touch would have caused neuron S to release neurotransmitter from a small number of vesicles, leading to a weak gill withdrawal (as in [Figure 3.10B](#)). But now, this mild touch causes neuron S to release neurotransmitter from a larger number of vesicles ([Figure 3.11B](#)), so that M is more likely to fire, leading to a stronger gill-withdrawal response ([Brunelli, Castellucci, & Kandel, 1976](#); [Castellucci & Kandel, 1976](#)). In effect, the prior tail shock at T has put the *Aplysia* on alert, making it sensitive to a subsequent light touch.

The key to sensitization in *Aplysia* is that it is **heterosynaptic**, meaning it involves changes across several synapses, including synapses that were not activated by the sensitizing event. Because of this feature, a tail shock increases responses to any future stimulus. For example, if the experimenter next touched the upper mantle (activating sensory neuron U) instead of the siphon (neuron S), the same over-reaction (gill withdrawal) would occur (see [Figure 3.11](#)). As with habituation, the degree of sensitization depends on the strength of the initial stimulus: a single mild shock can produce sensitization that lasts for minutes; four or five shocks together can produce sensitization that lasts 2 or more days ([Marcus et al., 1988](#); [Squire & Kandel, 2000](#)).

heterosynaptic

Occurring in several nearby synapses simultaneously.

In applying the dual process theory to *Aplysia*, note that in this organism, the state system responsible for sensitization is not completely separate from the mechanism for habituation. Instead, interneurons that are part of the circuit that generates the reflexive response increase synaptic transmission. All the same, two independent processes are associated with habituation and sensitization effects—a decrease in glutamate in specific synapses and an increase in serotonin at many synapses, respectively—again, in keeping with the dual process theory of habituation, which thus does a good job of accounting for these effects in *Aplysia*. Dual process theory was originally developed to explain habituation in mammals, so the fact that it can also capture many of the aspects of habituation and sensitization in a marine invertebrate suggests that many different species learn from repetition in similar ways.

Past behavioral research has often treated habituation as “the opposite” of sensitization because of the opposing effects of stimulus-evoked responses in these two types of learning. Work with invertebrates has made it clear, however, that sensitization and habituation are better viewed as *interacting* learning mechanisms (as the dual process model proposes). In addition, studies of dishabituation in *Aplysia* show that the neural circuits that produce dishabituation also overlap with those that produce sensitization—and yet they are not identical ([Bristol, Sutton, & Carew, 2004](#)). On the other hand, research suggests that desensitization, which behaviorally is quite similar to habituation, is more similar to sensitization at the neural level in that it appears to be heterosynaptic ([Poon & Young, 2006](#)). Nevertheless, desensitization is not simply sensitization in reverse; it does not involve decreases in the release of serotonin. Instead, desensitization seems to involve active inhibition of synaptic transmission by an independent set of inhibitory neurons. Perhaps you, like Kandel at the start of his investigations, expected that something as seemingly basic as increases or decreases in the reflexive movements of an *Aplysia* encountering repetitive stimuli would be a relatively simple learning process to understand. Rather, the work on *Aplysia* shows that even “simple” learning processes can be quite complex.

TEST YOUR KNOWLEDGE

Synaptic Mechanisms of Learning in *Aplysia*

Habituation and sensitization result from different effects on synaptic transmission in *Aplysia*. For the following situations, see if you can deduce whether habituation or sensitization has occurred and what is happening in the stimulus–response pathway. ([Answers appear at the end of the chapter.](#))

1. Observations reveal that only one synapse, where once there were two, is now connecting a neuron that responds to stimulation of the tail to a neuron that contributes to the gill-withdrawal response.

2. Measurements of glutamate released around a motor neuron show that levels of glutamate are increasing over time.
3. Anatomical analyses of neural circuits reveal a larger number of synapses associated with the gill-withdrawal reflex circuit than are normally seen.
4. Recordings of motor neuron activity indicate that the neurons are firing less.

Perceptual Learning and Cortical Plasticity

When an *Aplysia* is learning about touches to parts of its body, its sensory neurons detect those touches the same way every time. Repeated touches change the strength of the connections between sensory neurons and motor neurons, but sensory processing itself does not change: the amount of neural firing generated by sensory neurons with each touch is stable throughout habituation. Habituation occurs when an *Aplysia* learns to ignore touches to its siphon. In contrast, perceptual learning involves increases in the ability to make distinctions between similar inputs. In perceptual learning, an individual learns to recognize stimulus differences that were not noticed before, and sensory neurons may change how they respond to events over time.

Cortical Maps of Sensations

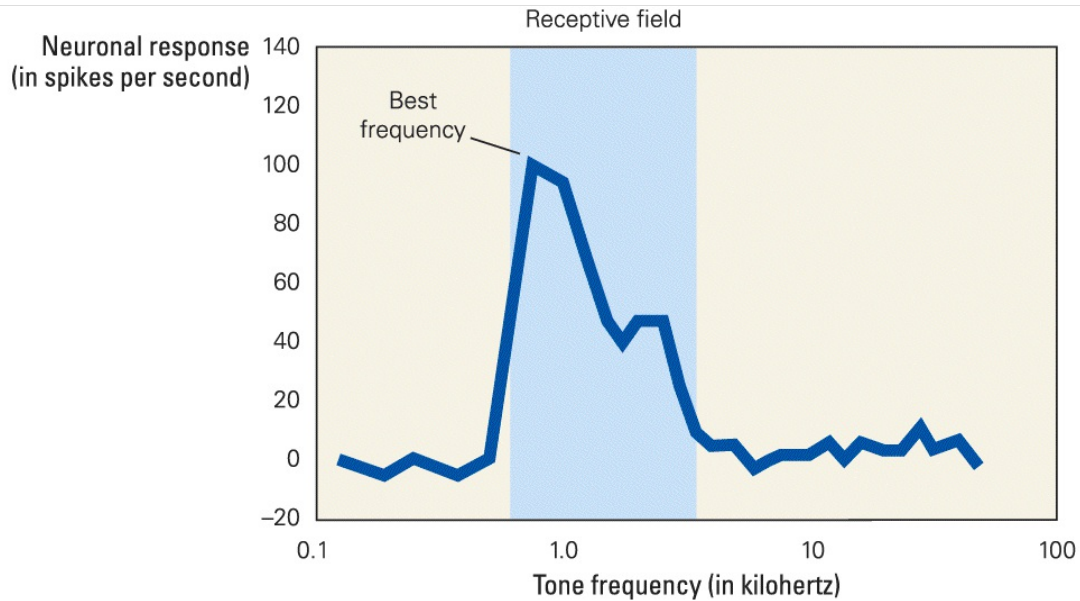
Unlike *Aplysia*, which have relatively simple neural circuits, humans and other mammals depend on the cerebral cortex to process information about stimuli and distinguish meaningful differences in their features. *Sensory cortices* are especially important for making such distinctions. As described in [Chapter 2](#), sensory cortices are areas of the cerebral cortex that process visual stimuli (sights), auditory stimuli (sounds), somatosensory stimuli (tactile feelings), and so on. Within each of these brain regions, individual neurons respond to different stimulus features. For example, some neurons in your auditory cortex fire strongly when you hear a high-pitched note, whereas others fire strongly when you hear a low-pitched note.



Image Source/DigitalVision/Getty Images

Mix engineers spend their lives combining different sounds to achieve musical balance. How might their auditory receptive fields differ from those of other people?

The range of sensory stimuli that cause a particular cortical neuron to fire is called the neuron's **receptive field**. [Figure 3.12](#) shows the receptive field for one neuron in the auditory cortex of a guinea pig, measured using single-cell recording techniques (described in [Chapter 2](#)). The neuron fired when the guinea pig heard tones pitched between 0.7 and 3 kilohertz (kHz); this range of pitches is the neuron's receptive field. The neuron fired most when the guinea pig heard tones pitched near 0.9 kHz. Neuroscientists would say that this cortical neuron is *tuned* to 0.9 kHz, meaning that this pitch causes the most firing.



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FIGURE 3.12 Receptive field of a neuron in the auditory cortex of a guinea pig Receptive fields are identified by measuring the amount of neural activity produced in response to different stimuli—in this case, to sounds ranging from 0.1 to 100 kilohertz (kHz). This neuron responds most to 0.9 kHz, but it also responds to a narrow range of similarly pitched sounds, and this range constitutes the neuron’s receptive field.

Data from [Weinberger, 2004](#).

Description

The horizontal axis shows Tone frequency in Kilohertz. The vertical axis shows the Neuronal response in spikes per second. The data in the graph are as follows. It starts from 0 and slightly declines to negative 10 on the vertical axis. Thereon, it gradually inclines and reaches a point 100 on the vertical axis. A label pointing to 100 reads, Best frequency. Further, it declines to a point, 40 on the vertical axis. Later, it gradually decreases and moves in small peaks. Finally, it reaches a point from 10 to 100 on the horizontal axis. All data are appropriate.

The shaded region between 0.8 and 5.0 on the horizontal axis is labeled Receptive field.

receptive field

The range (or “field”) of physical stimuli that activate a single neuron.

In the case of touch, sensory receptors in your skin send signals up your spinal cord, through your thalamus, and into your somatosensory cortex, a region introduced in [Chapter 2](#) and shown in [Figure 2.8](#). [Chapter 2](#) also introduces the motor cortex and describes how it contains a map, called a *homunculus*, of different regions of the body that it controls ([Figure 2.14](#)). The somatosensory cortex has a similar homunculus that maps parts of the body receiving touch messages ([Figure 3.13](#)). Thus, whenever you touch something with your index finger, a specific

set of neurons within your somatosensory cortex becomes active, and you not only perceive that your finger has contacted something, you perceive what the surface that you have touched feels like.

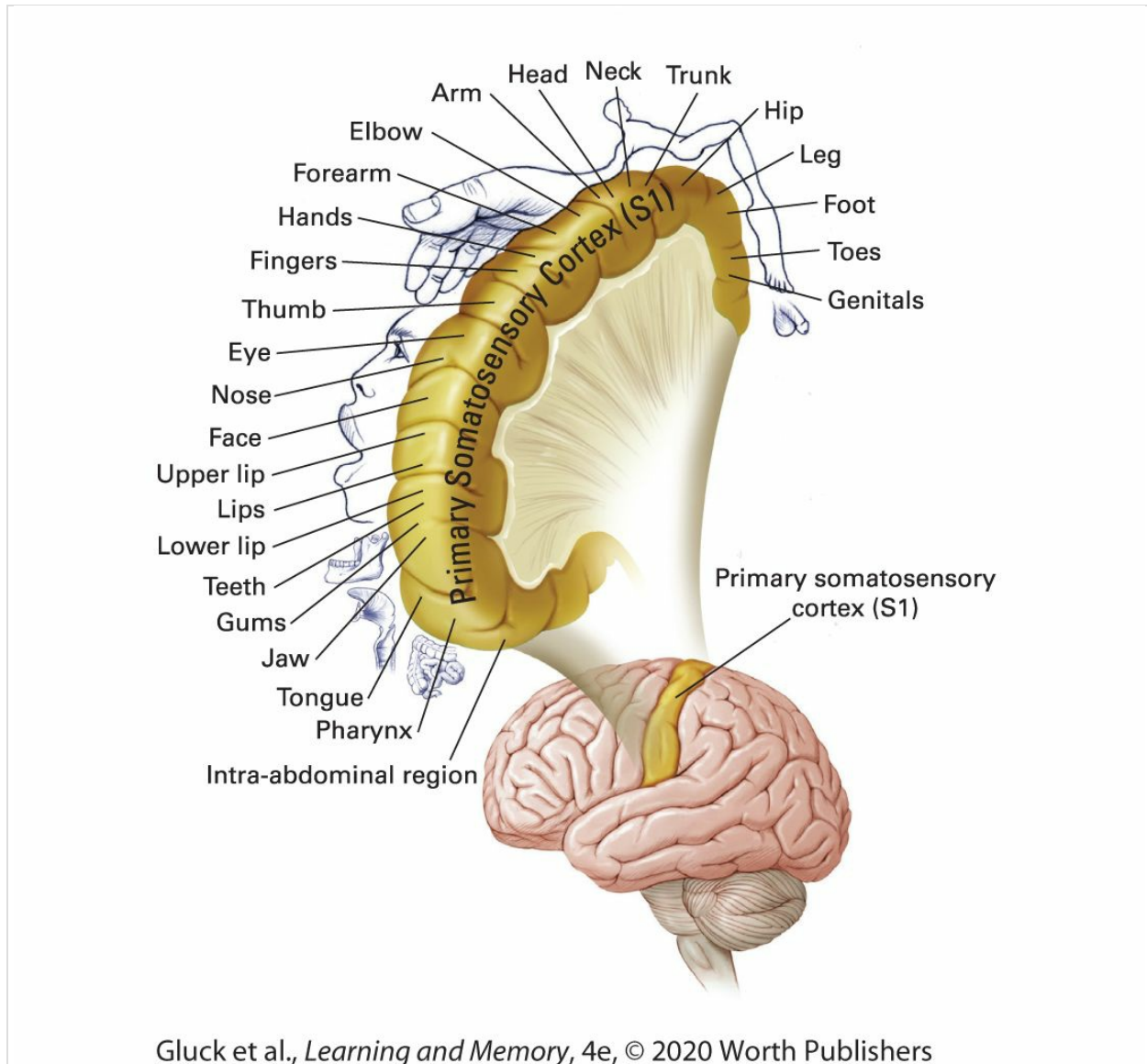


FIGURE 3.13 The homunculus corresponding to the human somatosensory cortex Different regions of the somatosensory cortex respond most strongly to touches of specific body parts. These regions are organized such that body parts close together activate adjacent regions of the somatosensory cortex.

Description

A region on the forebrain is labeled, Primary somatosensory cortex (S1). An enlarged view of the somatosensory regions is labeled in an anti-clockwise direction and are as follows. Genitals, Toes, Foot, Leg, Hip, Trunk, Neck, Head, Arm, Elbow, Forearm, Hands, Fingers, Thumb, Eye, Nose, Face, Upper lip, Lips, Lower lip, Teeth, Gums, Jaw,

In the somatosensory cortex, the receptive field of a neuron is defined as the patch of skin or other tissue that, when stimulated, causes the neuron to fire. In general, the more neurons that are tuned to a particular type, source, or strength of stimulus (or any other feature of a stimulus), the more capable the organism is of making fine distinctions related to that stimulus. So, for example, in the body map shown in [Figure 3.13](#), the region of cortex sensitive to touches on the thumb is larger than the region sensitive to touches on the neck; a larger region in the cortical map means more neurons are tuned to that part of the body, with the result that the skin on the thumb is able to detect finer distinctions than is the skin on the neck.

The spatial organization (body map) of the somatosensory cortex shown in [Figure 3.13](#) reveals that neurons with adjacent receptive fields are often found clustered close together. Visual and auditory cortices likewise contain clusters of similarly tuned neurons. For instance, recordings from the surface of a guinea pig's auditory cortex would reveal that adjacent neurons respond to gradually increasing or decreasing sound frequencies—that is, neurons tuned to 0.9 kHz are surrounded by neurons tuned to 0.8, 0.9, or 1.0 kHz. If you sat at a piano and played the keys one at a time from left to right up the keyboard, activity in your auditory cortex would gradually shift in a pattern corresponding to the movement of your hand across the keyboard.

For most of the history of neuroscience, it was thought that neurons in sensory cortices respond to sensations in ways that are directly analogous to how piano strings respond to the pressing of piano keys. Specifically, it was thought that every time a specific set of sensory receptors detects a stimulus, a particular set of cortical neurons becomes active. Neuroscientists were surprised to discover that in fact the receptive fields of neurons in sensory cortices change over time. The capacity for cortical receptive fields and cortical spatial organization to change as a result of experience is called [cortical plasticity](#).

cortical plasticity

The capacity to change cortical organization as a result of experience.

If what you perceive depends on how neurons in your sensory cortices are tuned, and if the tuning of your sensory cortices changes over time, then what does this suggest about your perception? It suggests that your perception may also change over time—which is exactly what

studies of perceptual learning show. The clearest evidence that repeated experiences can change perception by changing sensory cortices comes from studies of perceptual development.

Cortical Plasticity During Development

Normally, as young organisms develop, their ability to perceive differences in visual stimuli increases. Neurophysiological studies of neurons within the visual cortices of kittens show that their neurons become more selective over time and that maps in their visual cortex become more organized. If, however, a kitten's eye is sewn shut during development or if vision in an infant's eye is occluded by a cataract for several years, then the acuity of that eye will be permanently degraded, even if vision in that eye is later restored. (See [Chapter 12](#) for a more detailed discussion of *sensitive periods* during development.) Similarly, if young animals are experimentally deprived of vision in one eye, their cortical neurons will show less tuning to that eye than is seen in animals that grew up with both eyes fully functional. These findings suggest that normal development of visual cortical maps in mammals requires neural activity from both eyes. In other words, repeated visual experiences shape the organization of the visual cortex during development, which in turn determines an organism's perception of the visual world ([Morishita & Hensch, 2008](#)).

If perceptual experiences change how sensory cortices respond to stimuli, what happens if stimulation from both eyes is cut off, such as when a person is born blind or loses her sight soon after birth? Neuroimaging studies show that the areas of visual cortex that normally respond to visual stimuli in sighted people will, in blind people, respond to sounds and tactile stimulation. For example, activity in the visual cortex is seen to increase in blind individuals during Braille reading and other tactile tasks, but it decreases in sighted individuals performing these same tasks ([Lewis, Saenz, & Fine, 2010](#); [Sadato et al., 1998](#)).

Cortical plasticity produced by early blindness has been studied experimentally in developing opossums ([Kahn & Krubitzer, 2002](#); [Karlen, Kahn, & Krubitzer, 2006](#)). Researchers blinded half of the animals at birth and then, when the animals reached adulthood, exposed both the blinded and sighted opossums to visual, auditory, and somatosensory inputs to measure between-group differences in cortical structure and receptive fields. Sighted opossums possessed distinct cortical regions tuned exclusively either to visual, auditory, or somatosensory inputs. At the same time, receptive fields in other regions of the cortex were *multimodal*, meaning that neurons in those

areas responded to inputs from more than one sensory modality—for instance, visual *and* auditory stimuli. A different pattern was seen in opossums that had grown up blind. The cortical areas that were tuned exclusively to visual stimuli in sighted opossums had shrunk, and *within* those areas, some neurons now responded to auditory or somatosensory stimuli or both. In addition, the auditory and somatosensory areas of the cortex had increased beyond normal size. Most striking of all, the blinded opossums possessed a new cortical region with unique anatomical and physiological characteristics that did not exist in any sighted opossum’s brain.

Clearly, developmental experiences can have a huge effect on how neurons within sensory cortices respond to stimuli, influencing both the perception of sensory events and the development of responses to perceived events. In the case of opossums that developed without sight, the absence of vision radically changed the sensory experiences to which cortical neurons were exposed, and the opossums’ brains changed accordingly. In all animals, not just those that have been blinded (or similarly injured) at birth, experience modifies sensory cortical maps. Your own cortical maps changed drastically during your infancy, and they will continue to change throughout your life, although you will not perceive that this is happening. In [Section 3.3](#), we describe how perceptual learning makes it possible for individuals to use new technologies to overcome sensory deficits such as blindness and deafness.



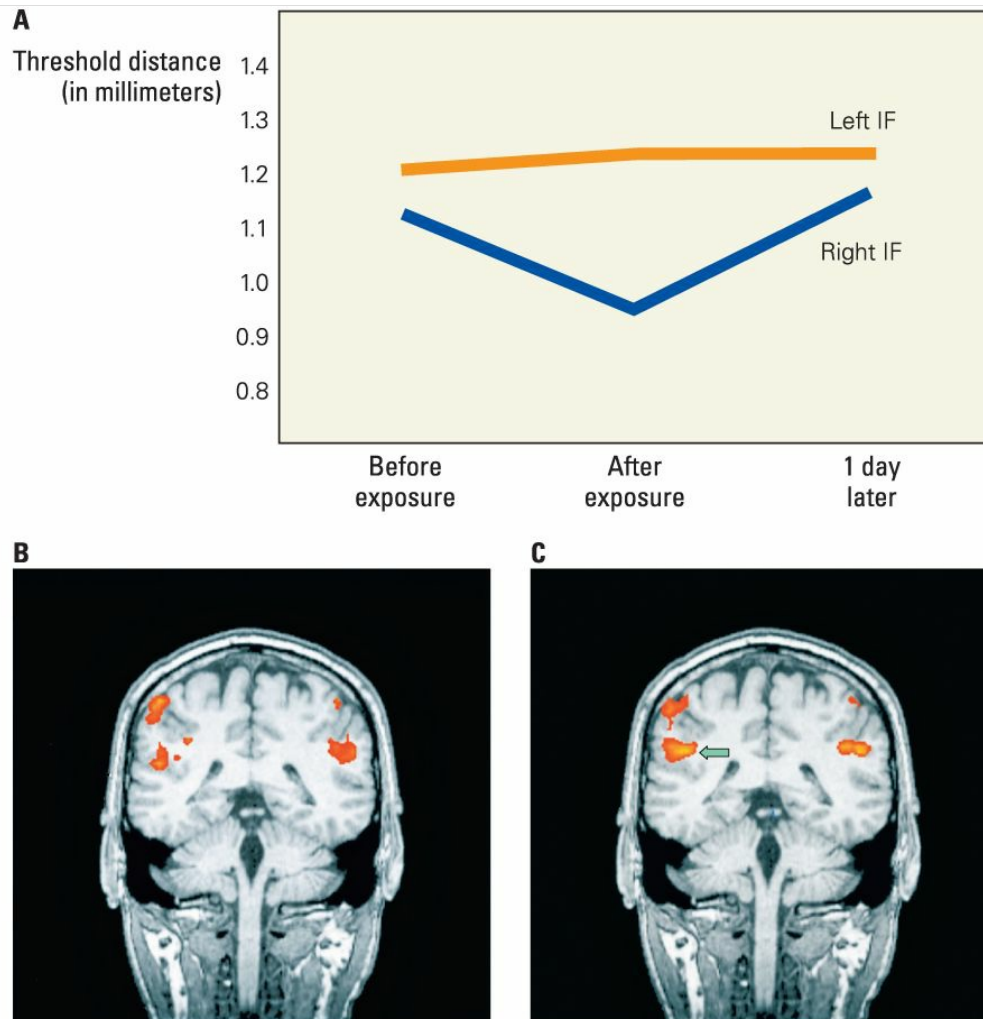
mawear/iStock/Getty Images

Studies with opossums have revealed that the development of cortical structure and function depends on repeated experiences. How might sensory cortices in an opossum born deaf as well as blind differ from those in animals born with all sensory modalities intact?

Cortical Changes in Adults After Exposure

During development, inputs from different sensory receptors determine how cortical neurons become tuned, as well as the proportion of available neurons that respond to a particular class of stimuli. Someone born without vision is likely to have proportionately more neurons available to respond to tactile stimuli, while someone born deaf typically will come to have larger cortical regions sensitive to visual stimuli. Granted, these are extreme cases of depriving a brain of a particular class of input. What might happen in more subtle cases—for example, in a person who chooses to listen only to rap music, classical music, or country music? Might the map in a person's auditory cortex change depending on the kinds of songs the person listens to the most? How much exposure is required for the cortical neurons to become retuned?

Neuroimaging studies suggest that it is relatively easy to retune neurons within the sensory cortices of adults; it can even happen in less than a day! For instance, simply touching a person's fingertip repeatedly with tiny pins was shown to improve the person's ability to distinguish subtle differences in the pins' positions. Initially, a person could discriminate two simultaneous touches on the tip of her index finger only when the touches were spaced at least 1.1 mm apart ([Figure 3.14A](#)). After receiving 2 hours of exposure consisting of repeated, simultaneous stimulation of two closely spaced points (0.25–3 mm apart) on the tip of the index finger, a participant's ability to discriminate touches improved, but only for the finger that experienced the repetition ([Dinse, Ragert, Pleger, Schwenkreis, & Tegenthoff, 2003](#); [Hodzic, Veit, Karim, Erb, & Godde, 2004](#); [Pilz, Veit, Braun, & Godde, 2004](#)).



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 Figures courtesy of Ben Godde, Jacobs University, Bremen, Germany

FIGURE 3.14 Cortical reorganization in humans after exposure (A) Participants improved their ability to distinguish two separate touch points on their right index finger (Right IF) after 2 hours of passive exposure to stimulation of closely spaced points on that finger. (B) fMRI showing cortical activation patterns in somatosensory cortex during tactile stimulation of the right index finger before exposure. (C) After 2 hours of stimulation to the right index finger, activation in the left hemisphere (where the right finger is represented) has increased.

Description

The horizontal axis shows three instances: Before exposure, After exposure, and 1 day later. The vertical axis shows Threshold distance in millimeters. The data in the graph are as follows. The line representing Left IF starts from (Before exposure, 1.2) and ends at (1 day after, 1.2). The line representing Right IF starts from (Before exposure, 1.1) and then gradually declines to (After exposure, 0.9). Further, it inclines to (1 day after, 1.1).

Two photos show the M R I scan report of the human brain. The first photo shows a few highlights on the left and the right side of the brain. The second photo also shows a few highlights on the left and the right side of the brain. An arrow on the left side of the brain points toward a bigger highlight on the left side.

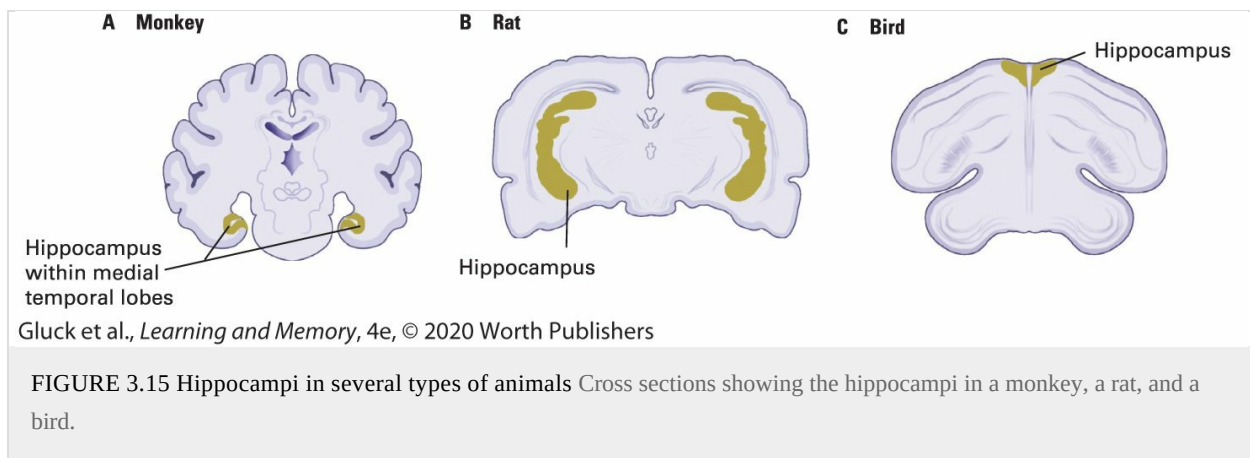
Like the experiment with scribbles, described earlier (see [Figure 3.6](#)), this study shows that humans can learn to make fine distinctions through mere repeated exposures. What is going on in the brain when this happens? Before repeated exposures, fMRI showed that touching the index finger resulted in localized activation within the somatosensory cortex ([Figure 3.14B](#)). After the same finger was stimulated repeatedly for 2 hours, subsequent instances of stimulation activated a larger region of the somatosensory cortex than was observed before exposure ([Figure 3.14C](#); [Hodzic et al., 2004](#)). Thus, repeated touching of the fingertip led to both perceptual learning and cortical reorganization. The increase in the size of the region of somatosensory cortex that was selectively activated during stimulation of the tip of the index finger was likely associated with an increase in the number of cortical neurons tuned to touches of the fingertip.

Many neuroscientists now believe that all forms of perceptual learning in mammals depend on cortical plasticity ([Dinse & Merzenich, 2002](#); [Kujala & Naatanen, 2010](#)). In fact, almost every type of learning discussed in the subsequent chapters of this book has been shown to lead to retuning of cortical receptive fields ([Hoffman & Logothetis, 2009](#); [Weinberger, 2007](#)). The role of cortical plasticity in learning and memory is further discussed in [Chapter 6](#), in relation to generalization, discrimination, and concept formation.

Hippocampal Involvement in Spatial Learning and Familiarity

As mammals experience repetitions of sensory events, neurons in their sensory cortices gradually become tuned to specific features of those events. Might similar processes, perhaps elsewhere in the brain, explain the kinds of latent learning observed when individuals actively explore the world around them? One way that researchers have tried to answer this question is by measuring and manipulating brain activity in rodents performing spatial tasks. Initially, the emphasis was on discovering where memories of mazes were stored in the cortex. Karl Lashley's failed attempt to locate cortical *engrams* for maze learning in rats (described in [Chapter 2](#)) represents one of the earliest efforts. Later neurophysiological studies of rats navigating through mazes revealed that spatial learning actually depends much more on activity in the hippocampus than on cortical engrams. What happens to neurons in the hippocampus as rats explore their environment?

In humans and other primates, the hippocampus is a relatively small structure that lies just beneath each temporal lobe (see [Figure 2.3](#) and [Figure 3.15A](#)). In rodents, however, the hippocampus makes up a much larger proportion of the brain ([Figure 3.15B](#)). Many other vertebrates, including birds and reptiles, also possess a hippocampus. Knowing that the hippocampus has a role in navigation and spatial memory, and generalizing from anatomical differences between species, you might expect that rats, with their large hippocampus, should be quite adept at spatial learning. This prediction is borne out by the success with which rats learned to traverse thousands of different mazes during the early history of psychological research.



Description

The first illustration shows a bilateral view of the hippocampus of a Monkey. Two tiny spots in the northern and southern hemispheres are labeled, Hippocampus within medial temporal lobes.

The second illustration shows a bilateral view of the hippocampus of a Rat. Horn-shape areas in the northern and southern hemispheres are labeled, Hippocampus.

The third illustration shows a bilateral view of the hippocampus of a bird. Tiny curved tube-like structure on the tip of the northern and southern hemisphere is labeled, Hippocampus.

Identifying Places

As a first step toward understanding the role of the hippocampus in spatial learning, English neuroscientist John O'Keefe implanted electrodes in rats' hippocampal regions to record neuronal activity under various conditions ([O'Keefe & Dostrovsky, 1971](#)). When the rats were placed in an environment and allowed to explore freely, the investigators made a surprising discovery. Some hippocampal neurons seemed to fire only when a rat wandered into particular locations, and other hippocampal neurons fired only when the rat was in other locations. O'Keefe

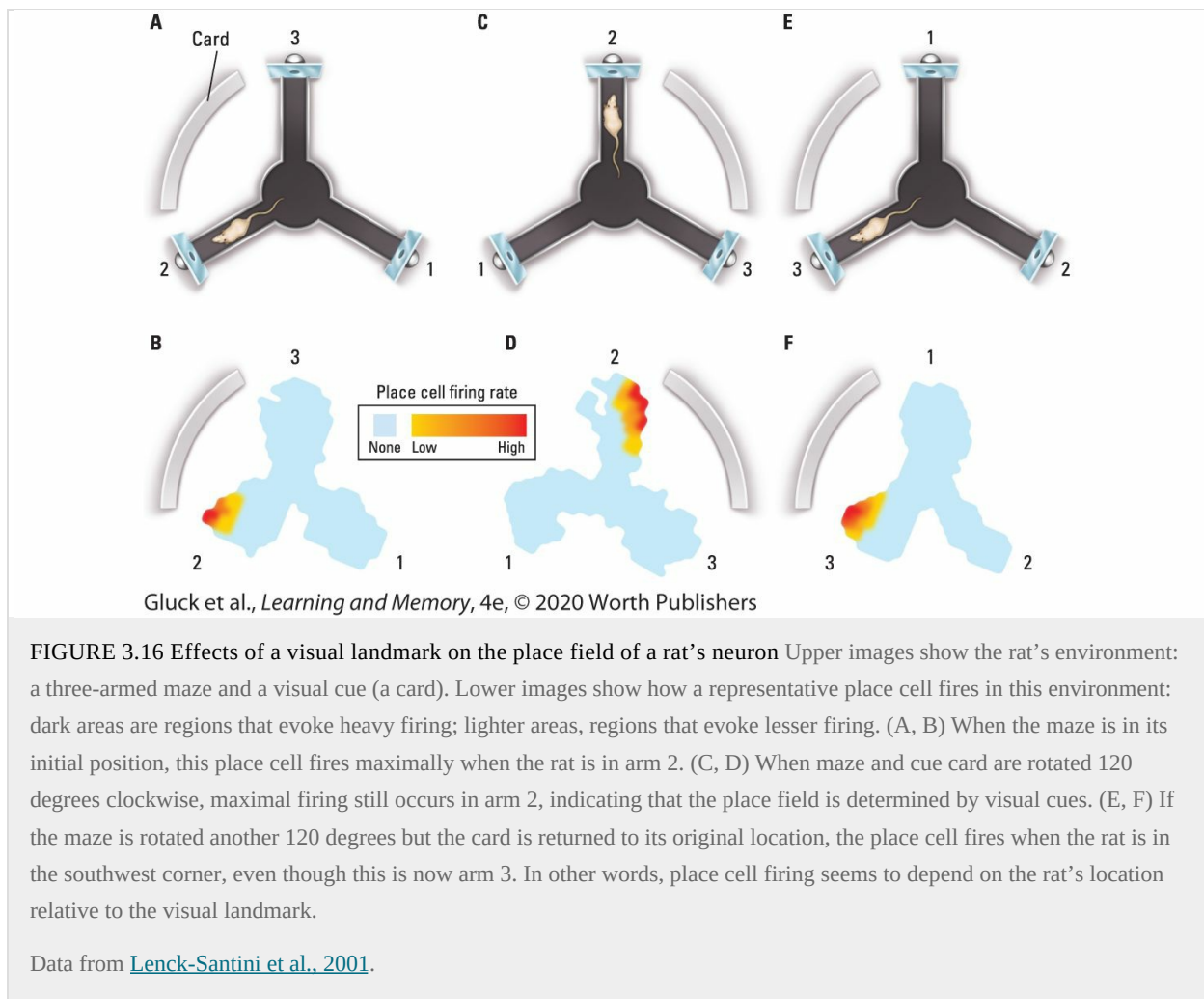
coined the term [place cells](#) to refer to neurons with such spatially tuned firing patterns. Each of these neurons has a certain preferred location to which it responds with maximal activity, and this location was termed the *place field* for that neuron (analogous to the receptive fields of sensory cortical neurons described earlier). O’Keefe suggested that place cells might form the basis for spatial learning and navigation—and in 2014, he received a Nobel Prize for his contributions to this groundbreaking field of research.

place cell

A neuron that fires maximally when the organism enters a particular location within an environment.

How might place cells help with spatial navigation? If a certain neuron fires only when an individual is in a particular place, then that neuron might serve as an identifier for that place (much like a road sign at a street corner or a mile marker along the highway). When the neuron fires, the brain then “knows” that the body is in a particular location. A person might figure out where he or she is just by noting which place cell is firing.

Part of what leads a place cell to respond seems to be the animal’s inner sense of its location in space: a rat’s place cells often continue to respond in an orderly fashion even when the rat is running through a maze with the lights out. But place cell responses also depend heavily on visual inputs. For example, suppose a rat is allowed to explore a maze like the one shown in [Figure 3.16A](#). This maze has three identical arms (labeled 1, 2, and 3 in the figure) and one salient visual cue: a card placed outside the maze between arms 2 and 3 that is visible over the side. After a rat first explores the maze, various place cells in its hippocampus will respond to specific parts of this maze. One hippocampal neuron, for instance, had the place field shown in [Figure 3.16B](#) (darker areas indicate maximal firing; lighter areas, lesser firing). In other words, this place cell responded the most when the rat was at the outer edge of arm 2, on the side nearest the card ([Lenck-Santini, Save, & Poucet, 2001](#)). This is the southwest corner of the maze as oriented in [Figure 3.16A](#). (Other place cells, not shown, responded maximally when the rat was in other locations in the maze, such as the center of the maze, or halfway down arm 1, or at the northeast end of arm 3.)



Description

Six illustrations are shown. They are labeled as A, B, C, D, E, and F. The group of the first three illustrations, labeled A, C, and E, are a three-armed maze. Each arm of the three-armed maze is labeled 1, 2, and 3. The second three illustrations labeled B, D, and F are illustrations of place cell fires in this environment. Each arm of the illustration that represents place cell fires in this environment is labeled 1, 2, and 3. The legend in between the illustrations shows the place cell firing rate as normal, low, and high. The first illustration A of the three-armed maze has a card placed between 2 and 3 or the left of the maze. The rat is shown moving toward the second arm of the maze on the left. Illustration B explains the place cell firing rate for the first maze. The firing rate is low and high toward the southwestern region of the illustration labeled 2. The areas labeled 1 and 3 appear normal.

The second illustration B of the three-armed maze has the card placed between 2 and 3 or the right of the maze. The rat is shown moving toward the second arm on top. Illustration D explains the place cell firing rate for the second maze. The firing rate is low and high toward the northeastern region of the illustration labeled 2. The areas labeled 1, and 3 below appear normal.

The third illustration C of the three-armed maze has the card placed between 1 and 3 or the left of the maze. The rate is shown moving toward the third arm on the left. Illustration E explains the place cell firing rate for the third maze. The

firing rate is low and high toward the southwestern region of the illustration labeled 3. The areas labeled 1 and 3 on the top and the right of the illustration appear normal.

Now suppose the experimenter takes the rat out of the maze and rotates the maze and card 120 degrees clockwise, as shown in [Figure 3.16C](#). What do you think will happen when the rat is put back in the maze ([Figure 3.16C](#))? Will the place cell shown in [Figure 3.16B](#) continue to fire whenever the rat is in the southwest corner of the maze? Or will it fire instead when the rat is at the end of arm 2, even though that is now the north-most corner of the maze? The answer is shown in [Figure 3.16D](#): the place cell's preferred location has rotated along with the maze. In this particular case, since the three arms all look, smell, and feel similar, the rat probably used the visual cue (card) as a landmark. To confirm this idea, the experimenter rotated the maze again, another 120 degrees clockwise, but the card was returned to its original place, to the west ([Figure 3.16E](#)). Under these conditions, the place cell again fired in the southwest corner of the maze, even though this was now arm 3 ([Figure 3.16F](#)). These findings illustrate the importance of visual landmarks (such as the card) in determining when a hippocampal place cell will fire.

Some place cells have place fields that are stable for months: if the rat is returned to the maze in [Figure 3.16](#) after a long absence, the same place cell may still fire when the rat is in the same location as before. The stability of place fields and their selectivity in terms of particular visual scenes are consistent with the idea that place cells provide the basis for a “cognitive map” that rats use to navigate the world. But how, exactly, do place cells become tuned to a particular place?

One factor affecting the creation of place fields is experience. When rats repeatedly explore an environment, their place cells gradually become more sensitive to locations within that environment ([Lever, Wills, Cacucci, Burgess, & O'Keefe, 2002](#)). Imagine the size of the place field in [Figure 3.16](#) (parts B, D, and F) getting smaller and smaller, providing an increasingly precise and reliable report of where in the maze the rat is. This place-field shrinkage seems to correlate with rats' spatial navigation abilities in a maze; experiments in which rats' place-field shrinkage is disrupted (e.g., by blocking inputs from the thalamus) show that the rats' spatial learning abilities decline ([Cooper & Mizumori, 2001](#); [Mizumori, Miya, & Ward, 1994](#); [Rotenberg, Abel, Hawkins, Kandel, & Muller, 2000](#)).

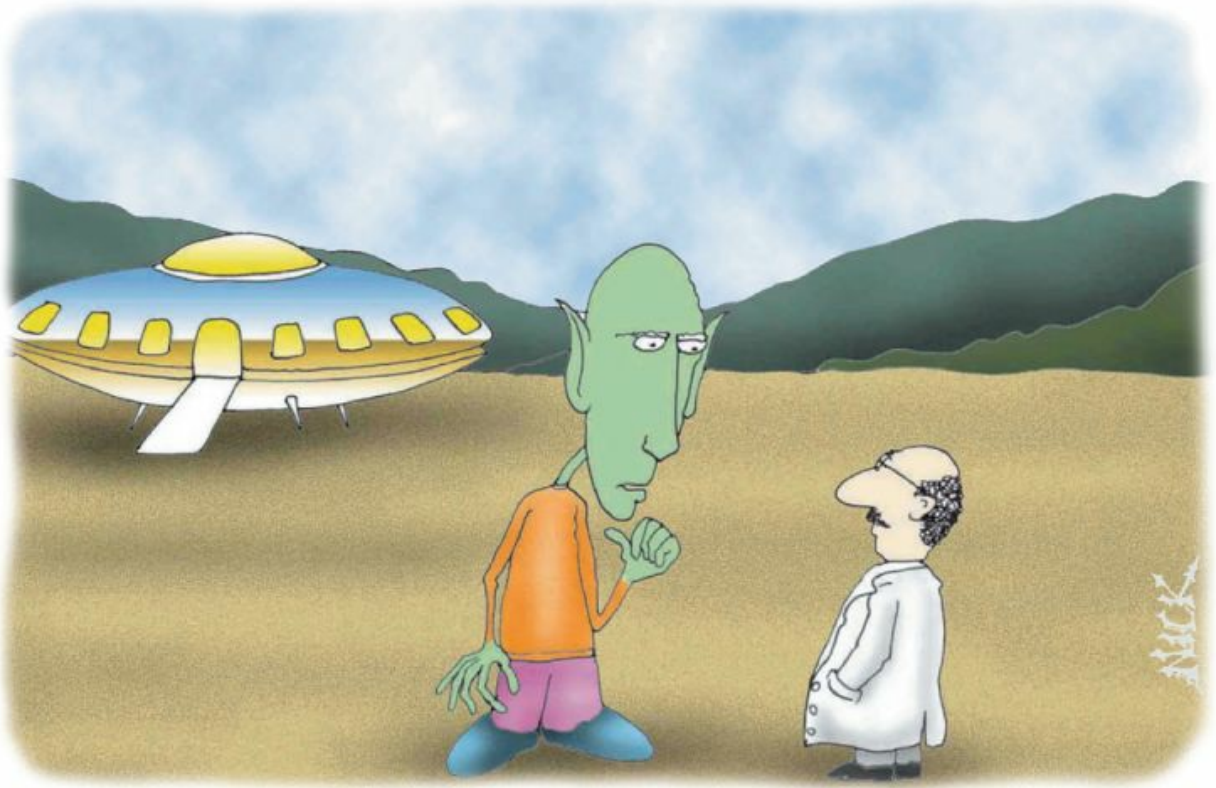
Place cells are not the only neurons tuned to spatial features. Other neurons located in cortical regions surrounding the hippocampus show selectivity when an animal is headed in a particular direction (*head direction cells*), or when animals cross through equal distances within an environment (*grid cells*). These neurons likely contribute to an individual's ability to learn about locations in ways that are similar to place cells.

These findings suggest that spatial learning is associated with changes in the activity of hippocampal neurons ([Rosenzweig, Redish, McNaughton, & Barnes, 2003](#)). This phenomenon may be related to the latent learning by which Tolman's rats came to know the layout of a complex maze even without food rewards. Perhaps as the nonrewarded rats explored the maze, their place fields shrank as much as (or more than) those of the rewarded rats, providing them with a precise representation of various locations within the maze.

Recognizing Familiar Objects

In order to use visual landmarks to navigate within a maze, rats must be able to recognize those landmarks. This suggests that as rats explore an environment, they not only learn how to navigate that environment, they also learn the properties of objects within and around the paths they are traveling. Recall that some of the earliest evidence of visual perceptual learning came from studies in which triangles and circles were mounted in the home cages of rats. If neurons within the hippocampus are sensitive to visual landmarks within a maze, might they also contribute to a rat's ability to recognize novel objects and distinguish them from familiar landmarks?

Several studies have examined this possibility in rats performing the novel object recognition task described earlier ([Cohen & Stackman, 2015](#)), as well as in monkeys performing shape recognition tasks ([Nemanic, Alvarado, & Bachevalier, 2004](#)). The results of these studies are mixed: some show deficits in object recognition after hippocampal lesions, while others show no effect. Cortical neurons in regions that send inputs to the hippocampus respond less to familiar objects than to novel ones, suggesting that they might play important roles in distinguishing familiar objects from objects that are unfamiliar ([Xiang & Brown, 1998](#)).



*"That? No, that isn't a U.F.O. It's on the ground now,
and you've identified it. That makes it an I.G.O."*

Nick Kim/Cartoonstock.com

Which of your brain regions would likely be engaged by an alien encounter?

Neuropsychological studies of patients with lesions to the hippocampus and surrounding cortical regions found impaired recognition of familiar objects ([Squire, Zola-Morgan, & Clark, 2007](#)).

Neuroimaging studies of healthy volunteers also implicate regions in the temporal lobes (near the hippocampus) in the detection of familiarity and novelty, although there continues to be debate about the specific roles that the hippocampus and cortical regions play. Intriguingly, electrical stimulation in cortical regions within the temporal lobes can produce feelings of *déjà vu* in human patients, further implicating these regions in the detection of familiarity ([Bartolomei et al., 2012](#)).

In addition, past findings suggest that neurons in the hippocampus and surrounding cortical regions contribute to exploratory learning in ways that go beyond simply constructing spatial

maps or identifying familiar objects. At the same time, other brain regions, including sensory cortices, may also play a pivotal role in object recognition. For example, changes in primary visual cortex (V1) activity are linked to both long-term habituation and to the detection of novel images ([Cooke, Komorowski, Kaplan, Gavornik, & Bear, 2015](#)). These and other findings suggest that the mechanisms of habituation, perceptual learning, and novel object recognition may be more closely linked than researchers previously thought. As you will see in the following section, such discoveries not only can provide new insights into the processes underlying various forms of learning but can also lead to new approaches to correcting those processes when they are disrupted by brain disorders.

Interim Summary

- In *Aplysia*, habituation is the result of synaptic depression in circuits that link sensory neurons to motor neurons. Long-term habituation involves physical changes in the connections between these neurons.
- Sensitization in *Aplysia* reflects heterosynaptic increases in synaptic transmission caused by the activation of interneurons.
- During perceptual learning, cortical neurons refine their responses to sensory inputs as discrimination abilities improve.
- Hippocampal place cells are neurons that become most active when an animal is at a particular location. These neurons and other spatially sensitive cells in surrounding cortical regions may help animals learn to navigate.
- Neurons in the hippocampus and surrounding cortical regions also contribute to object recognition, giving rise to a sense of familiarity.

3.3 Clinical Perspectives

Even though you probably are not consciously aware of it, habituation, sensitization, and familiarization influence every experience you have. From your ability to understand speech to your ability to find your way to school or work, everything you perceive is influenced by the memories you have acquired through repeatedly experiencing similar stimuli. When neural circuits that process sensory information are damaged or when highly aversive events are experienced, the result can be a fundamental change in perceiving, processing, and learning about stimuli, which in turn can affect mental health and quality of life.

Rehabilitation After Stroke: Habituation Gone Awry

The leading cause of brain damage in the United States is stroke. A [stroke](#) occurs when blood flow to some region of the brain stops or when an artery bursts. Lack of blood and swelling of brain tissue cause neurons in the affected region to die, effectively creating a brain lesion.

stroke

An event in which blood flow to some region of the brain stops or in which an artery ruptures, causing neurons in the affected region to die.

Immediately after a stroke, a patient often experiences large losses in perceptual function. For example, a patient may lose all sensation in one of his arms. Subsequently, although nothing may be wrong with the motor control of that arm, the patient may begin to ignore the numb arm and make greater use of the arm he can still feel. Over time, he may stop trying to use the numb arm altogether, a phenomenon called *learned non-use*. Basically, the patient gets used to the lack of feeling in the dysfunctional arm and gets in the habit (through repetition) of depending on the other arm.

Monkeys show similar patterns of learned non-use when they lose sensation in a limb. For instance, if somatosensory information from a monkey's left arm is blocked so that the monkey cannot feel the arm, the monkey will stop using that arm and start using the right arm exclusively. If the right arm is bound so that the monkey can no longer use it, however, the monkey may begin using the numb left arm, even after not using it for several years. Once the monkey has become accustomed to using the left arm again, releasing the right arm will typically

result in the monkey using both arms, showing that the monkey can overcome learned non-use of the dysfunctional arm ([Knapp, Taub, & Berman, 1963](#)). One interpretation of this functional recovery is that binding the right arm created a new situation that dishabituated the monkey's responses to numbness in the left arm.

Techniques like those used to overcome learned non-use in monkeys are also sometimes used in therapy for human stroke patients. For example, a patient who has lost the use of his left arm might consent to have his preferred right arm immobilized in a sling so that he is forced to try to use his left arm for eating, dressing, and other daily activities. As [Figure 3.17A](#) shows, patients who undergo this kind of therapy, called **constraint-induced movement therapy**, often recover much more function in their affected arm than do patients who are simply told to try to use their affected arm as often as possible ([Taub, Uswatte, & Elbert, 2002](#); [Wolf et al., 2010](#)).

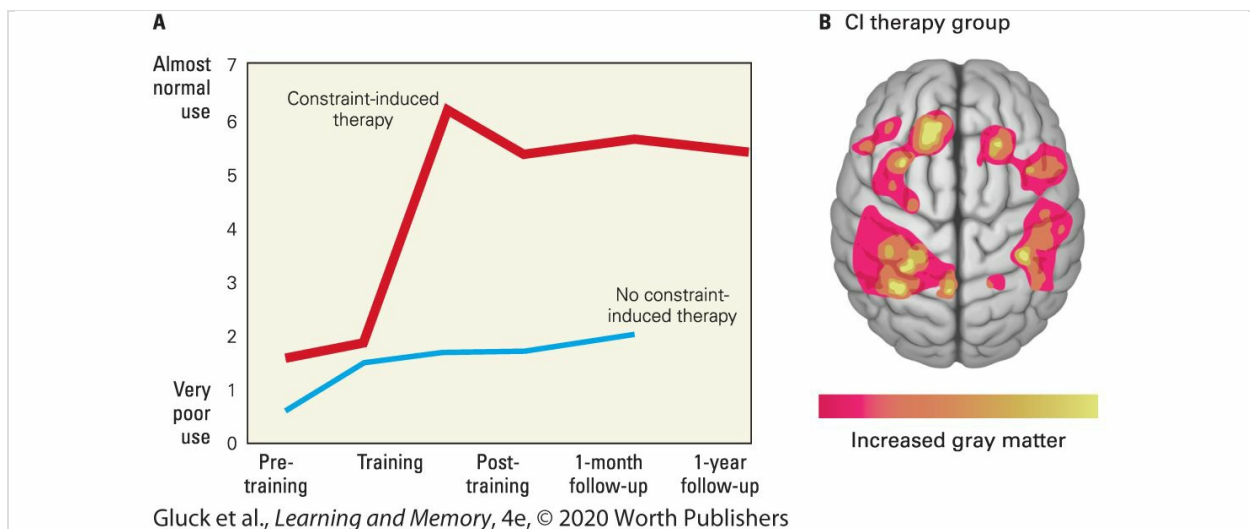


FIGURE 3.17 Overcoming learned non-use (A) Patients with cortical lesions affecting one limb (e.g., an arm) often start using the unaffected limb in preference—a learned non-use of the affected limb. The graph shows results for two groups of patients who initially (pretraining) had very little use of one arm. One group underwent constraint-induced movement therapy, which forced them to use the affected arm for daily activities. These patients showed dramatic improvements in function of the injured arm compared with control patients not receiving this therapy. (B) Increases in gray matter are observed after constraint-induced therapy (CI therapy group), but not in the comparison group.

Data from [Taub et al., 2002](#), and from Plastic Structural Brain Changes Produced by Different Motor Therapies After Stroke, [Gauthier et al., March 6, 2008](#).

Description

The horizontal axis plots Pre-training, Training, Post-training, 1-month follow-up, and 1-year follow-up. The vertical axis plots values from 0 to 7, in an increment of 1 unit. The scale from 0 to 1 is labeled Very poor use and from 6 to 7 is

labeled Almost normal use. The data in the graph are as follows. Two curved lines are shown. The curved line, No constraint-induced therapy starts from the range of 0.4 during pre-training. It slightly raises and reaches 2 at the 1-month follow-up. The curved line, Constraint-induced therapy starts from the range 1.5 during Pre-training. It suddenly inclines and reaches at 6 during Training. Thereon, gradually moves to 5 at the 1-year follow-up. All data are approximate.

The illustration on the right labeled C I therapy group shows the cross-sectional view of the human brain, with the gray matter highlighted. It is spread evenly through the northern and southern hemispheres of the brain.

constraint-induced movement therapy

A motor-rehabilitation technique in which unaffected limbs are restrained to increase usage of dysfunctional limbs.

The idea behind constraint-induced movement therapy is to force the patient to use the affected arm as often as possible to encourage changes in the cortex that compensate for stroke-related damage. This rehabilitation strategy is similar in some ways to the method used to reorganize cortical processing in opossums during development, as described in [Section 3.2](#). Recall that the newborn opossums were blinded so that cortical areas normally devoted to visual processing were instead reallocated to other sensory modalities. In stroke patients, constraint-induced movement therapy may help undamaged cortical areas take up some of the work that damaged areas once performed.

Neuroimaging studies show that constraint-induced movement therapy is associated with increases in blood flow to the sensory and motor cortex ([Wittenberg & Schaechter, 2009](#)). After this therapy, structural changes are seen in these cortical regions that are not seen after other rehabilitation techniques ([Figure 3.17B](#)). The beneficial effects of this therapy are thought to involve perceptual learning in which, through repeated efforts, patients acquire the ability to make distinctions between sensations that are initially quite faint (and therefore difficult to discriminate). Recall that weak inputs facilitate habituation. In the case of learned non-use, processes of habituation increase the difficulties patients face in overcoming physical limitations. Conversely, perceptual learning processes may help patients counteract reduced responses to weak stimuli by reorganizing the cortical regions that process those stimuli.

Sensitization to Stress in Anxiety and Depression

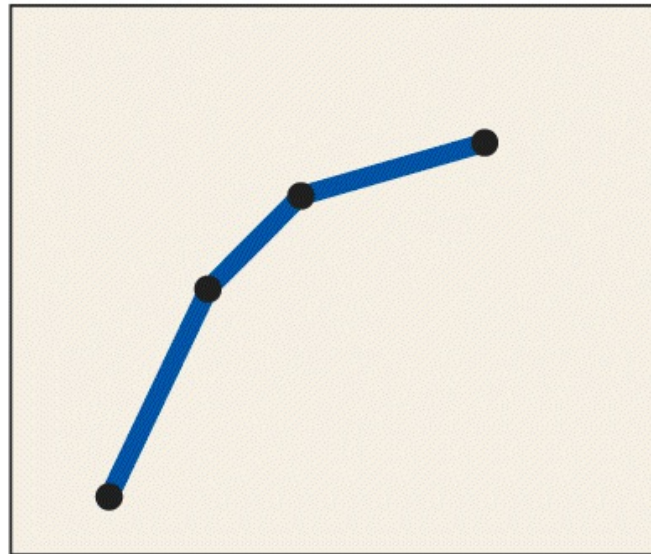
At repeated intervals in horror movies, directors use techniques intended to increase the viewers'

arousal, thereby increasing the likelihood and amplitude of startle reflexes evoked by scary and even not-so-scary stimuli, such as jumpy cats. In research laboratories and movie theaters, sensitization is relatively innocuous because the stimuli that lead to sensitization are purposefully controlled so as not to be too arousing. In some situations, however, the arousal that an organism experiences can be extreme and can have much more dire consequences. In the most severe cases, a single highly emotional event can lead to lifelong amplification of emotional responses to a wide range of stimuli, as occurs in *posttraumatic stress disorder* (discussed in [Chapter 10](#)). Recent studies suggest that even in cases where highly arousing events are not traumatic, a person's susceptibility to certain mental illnesses can still increase ([Harkness, Hayden, & Lopez-Duran, 2015](#)).

Early hints that repetition of arousing events could lead to problematic behavioral states came from studies showing that rodents that were repeatedly exposed to relatively low doses of amphetamines (a stimulant drug) showed gradual increases in bizarre, stereotyped behaviors that persisted months after drug exposure ended ([Robinson & Becker, 1986](#)). Repeated exposures to low doses apparently sensitized the animals to the effects of the drug because the odd, stereotyped actions were the same as those seen in nonsensitized rodents given higher doses of the drug ([Figure 3.18](#)).

Stereotypy
rating

44
40
36
32



1 3 5 7 9 11

Number of injections

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FIGURE 3.18 Effects of repeated injections on stereotyped actions Rats receiving injections of amphetamine in low doses every 3 to 4 days show progressive increases in the stereotypy of their head and limb movements. Their responses to the drug are sensitized by repetition.

Data from [Robinson & Becker, 1986](#).

Description

The horizontal axis shows the Number of Injections. The vertical axis shows the Stereotypy rating. The data of the graph are as follows. The curve begins at (1, 31). It gradually inclines and rises to (3, 37), which thereon reaches a point (5, 41). Later, it rises to reach a point (9, 42).

The amphetamine-related sensitization seen in these rat studies attracted researchers' interest because patients diagnosed with *schizophrenia* (discussed in [Chapters 6](#) and [9](#)) who were being treated with amphetamines sometimes showed similar behavioral problems. As in rodents, sensitization to amphetamines in humans can have negative effects on behavior long after the drugs are no longer in use. The fact that repeated drug-induced arousal produced long-lasting sensitization in presumably nonschizophrenic rats led researchers to consider the possibility that repeated natural arousal might have similar effects in humans who do not have mental disorders. Evidence consistent with this possibility was first described in the early 1990s ([Post, 1992](#)).

[Robert Post \(1992\)](#) found that after an initial stressful event triggered a disorder such as depression, increasingly minor stressful events could trigger additional bouts of depression later on. He proposed that this tendency occurred because some individuals become sensitized to stress and its associated physiological states, much the same way that the rats given amphetamines became sensitized to the stimulating effects of the drug. More recent studies indicate that depressed individuals show stronger responses to minor stressors than do healthy individuals ([Wichers et al., 2009](#)). In addition, depression promotes repetitive thoughts about symptoms and negative events ([Ruscio et al., 2015](#)), potentially further sensitizing individuals to low-level stressors.

These findings might seem to imply that people should avoid stressful events as much as possible to minimize the possibility of sensitization to stress. However, animal research suggests that minimized exposure to stressful events may actually lead to *greater* sensitivity to future stressors ([Liu, 2015](#)) and that experience with moderate stress over time may increase an individual's resilience when faced with a high-stress event. If similar effects occur in humans, then this would suggest that overprotective parents who do everything in their power to prevent their children from suffering could in some cases be increasing the risk that their children will be more adversely affected whenever those children do encounter highly stressful events. At the other end of the spectrum, parents who repeatedly expose their children to highly stressful events may also be gradually increasing their children's risk for mental illnesses later in life ([Laurent, Gilliam, Wright, & Fisher, 2015](#)).

Sensitization to stress not only can contribute to depression but also is thought to be a factor in anxiety disorders ([McLaughlin, Conron, Koenen, & Gilman, 2010](#); [Rosen & Schulkin, 1998](#)). Repeated exposures to high stress levels during development, for example, can increase chances for depression later in life and also puts children at risk of developing anxiety disorders, such as *obsessive compulsive disorder*. Just as depressed individuals tend to dwell on the factors that they perceive as contributing to their depressed state, individuals with obsessive compulsive disorder tend to repeatedly focus on certain thoughts that lead them to repeat certain actions many times. Again, such repetition may facilitate further sensitization, exacerbating the problem.

Pathological anxiety can be viewed as an exaggerated response to potentially scary stimuli, resulting when sensitization to stress associated with fear-inducing situations amplifies a

person's emotional responses to lower-level stimulation. (The role of learning in fear responses is discussed in detail in [Chapter 10](#).) Understanding the mechanisms that lead to sensitization is key to developing new techniques for preventing such disorders and for treating them effectively when they do arise.

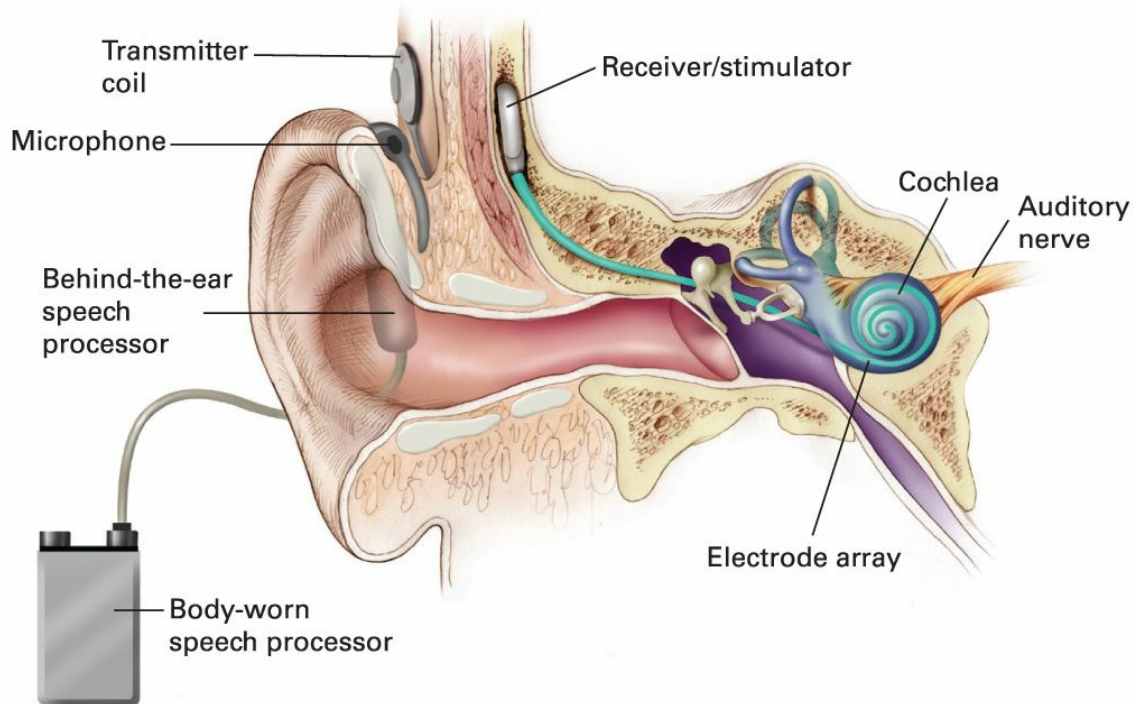
Human–Machine Interfaces: Regaining Sensory Modalities Through Perceptual Learning

If training can lead to cortical change that helps people recover motor functions after a brain injury, might it also enable people to develop perceptual abilities they don't have? For example, could perceptual learning help someone who is deaf learn to hear or help someone who is blind learn to see? This is indeed possible and has in fact already been accomplished with [sensory prostheses](#), which are electromechanical devices that interface with neural circuits that normally process sensory information.

sensory prosthesis

A mechanical device designed to supplement or substitute for a faulty sensory modality such as vision or hearing; the device's sensory detectors interface with brain areas that normally process those sensory inputs.

To date, the most extensively developed and successful sensory prosthetic technology is the [cochlear implant](#) ([Figure 3.19](#)). This device electrically stimulates auditory nerves to produce hearing sensations in profoundly deaf individuals, primarily to assist them in processing speech. Multiple electrodes implanted in the cochlea (the part of the ear that converts sounds into neural firing patterns) modify responses in the auditory nerve in ways that roughly simulate the neural activity normally produced by sounds. This technology is most effective in young children and in adults who have only recently lost their hearing. Conventional hearing aids amplify external sounds, but cochlear implants re-create the effects of sounds within the brain, generating “virtual sounds” from information about environmental sound that has been electronically detected and processed.



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FIGURE 3.19 Cochlear implant Cochlear implants use electricity to stimulate neurons in the auditory system, thereby creating virtual speech sounds in the brain.

Information from [Clarke, 2002](#).

Description

The implant consists of an outer portion that sits behind the ear and a second portion that is surgically fixed beneath the skin. The parts of the cochlear implant are labeled as follows. A microphone behind the ears aid in picking the sound from the environment. A behind-the-ear speech processor is present inside the ear, which is connected to a processor labeled Body-worn speech processor. It selects and arranges sounds picked up by the microphone. The small bead-shaped structure labeled Transmitter coil and receiver/stimulator accept signals from the speech processor. An electrode array is marked adjacent to spiral-shaped Cochlea. Both Cochlea and electrode array lie in the pathway of the auditory nerve.

cochlear implant

A sensory prosthesis that directly stimulates auditory nerves to produce hearing sensations in deaf individuals.

The virtual speech sounds generated by cochlear implants are quite different from normal speech, so people using the implants must learn to discriminate between the new sounds before they can begin to understand what they hear—an example of perceptual learning. Speech perception by individuals with cochlear implants shows initial rapid improvement in the early

months of use, followed by more gradual improvement over the ensuing years ([Clarke, 2002](#); [Tajudeen, Waltzman, Jethanamest, & Svirsky, 2010](#)). Although cochlear implants make it possible for those who are deaf to understand speech, they do not totally restore hearing. For instance, music is dramatically distorted by cochlear implants because the implants filter out many of the pitches that make music melodic.

It is likely that changes in speech-processing abilities after installation of a cochlear implant are the result of cortical plasticity, but this has yet to be experimentally demonstrated in humans. Neural activity generated by a cochlear implant may lead to changes in many areas of cortex because the implant provides the brain with access not only to new sounds but also to a range of other experiences that require the ability to hear (such as the opportunity to engage in spoken conversations). In cats, researchers have found that cochlear implants lead to massive changes in auditory cortex ([Klinke, Kral, Heid, Tillein, & Hartmann, 1999](#)). After implantation, auditory cortex becomes organized differently from what is seen in deaf cats without implants and also from what is seen in hearing cats, suggesting that the virtual sounds these cats hear are driving the observed changes in cortex.



Second Sight Medical Products, Inc.

Researchers at Duke University have developed retinal implants that are providing blind users with virtual sight. If these sensory prosthetics were used on an opossum blinded at birth, do you think the animal's sensory cortices would reorganize to be more like those of animals that had never been blind?

Most current sensory prostheses are designed to replace lost abilities, but in principle, it should also be possible to use such devices to enhance existing capabilities or to create new sensory abilities. In fact, researchers have already achieved this in rats: signals generated by head-mounted infrared sensors are sent directly to the rats' somatosensory cortex, giving them night vision ([Thompson, Carra, & Nicolelis, 2013](#)). No one knows how well cortical neurons in humans would be able to process inputs from sensors detecting stimuli such as infrared light or ultrasonic sounds that humans are normally unable to perceive. Given how easily deaf people have learned to process novel inputs from cochlear implants, however, it seems likely that the human brain could accommodate a wide range of technologically provided inputs through perceptual learning.

Interim Summary

- Learned non-use of a limb, which may be due to habituation, can be overcome by constraint-induced movement therapy, which forces the individual to use the affected limb.
- Repeated exposure to highly stressful events may sensitize a person to stress, which can increase that person's likelihood of developing depression or anxiety disorders later in life; the absence of exposure to stress can also be problematic.
- Sensory prostheses provide deaf or blind individuals with new sensory-processing capabilities. Use of these devices leads to perceptual learning that improves the user's ability to recognize sensory events.

Synthesis

Habituation, sensitization, perceptual learning, and other learning processes driven by repeated exposures to stimuli are often described as the simplest forms of learning. This is partly because even the most primitive animals (even amoebae!) show habituation. Moreover, these forms of learning demand little or no obvious effort from the learner, and they often occur without the learner's conscious awareness.

The processes involved in learning about repeated events can, however, be highly complex, drawing on the combined activities of multiple brain regions interacting in many different ways. A good example is the learning of landmarks for spatial navigation. Spatial learning can happen independently of observable changes in responses, which means it is difficult for an observer to determine what another individual is learning about any particular set of landmarks. If you see passengers staring out a car window as they ride along, you will be hard-pressed to tell if they are storing information about certain landmarks that might help them find their way back to that location later. In the window gazers' brains, complex combinations of events, including percepts both of visual patterns and of bodily movements, determine how neurons in the hippocampus and surrounding cortical regions respond, which in turn controls the nature and quality of the memories that are formed (a topic we explore in [Chapters 7](#) and [11](#)).

Repeated experiences can slow down your ability to learn (in the case of habituation) or speed it up (in the case of priming and perceptual learning). They can also affect your responses to other, seemingly unrelated, stimuli (as in the case of sensitization). For example, reading about the cannibalistic murderer Jeffrey Dahmer in the beginning of this chapter was probably novel in your experiences of reading psychology textbooks. If you have habituated to the relatively predictable material in most college textbooks (in other words, if you get bored reading textbooks), then this disturbing example may have caused your learned response to textbooks to dishabituate, which may have in turn increased your capacity to encode and remember the rest of the chapter, despite the fact that the discussions in it had little to do with psychopathic killers. How is it that repeated exposures to stimuli can generate such a wide range of learning phenomena? Part of the answer, at least in humans and other mammals, is the contribution made by the cerebral cortex, one of the most complex structures in the brain. Experience-dependent changes in how cortical neurons respond to repeated events constitute one of several powerful mechanisms that seem to contribute to perceptual learning, object recognition, spatial learning,

and habituation.

Changes in behavior that stem from memories of repeated events have important implications for our daily lives, especially when the brain is not processing information the way it should.

Understanding the plasticity mechanisms that enable brains to change with repeated experiences can help clinicians interpret the effects of cortical damage and take steps to alleviate sensory deficits. The ability of the brain to adapt in the ways described in this chapter may be the key to overcoming many mental disorders for which there are currently no cures. Thus, although the learning processes engaged by repetition can sometimes lead to negative outcomes (as in the circumstances surrounding Jeffrey Dahmer and in learned non-use), these processes also point to ways of rehabilitating patients and expanding people's perceptual abilities.

The situations and mechanisms that contribute to learning from exposure also play a role in other learning phenomena. For instance, in classical conditioning (discussed in [Chapter 4](#)), repeated exposure to a sequence of two events gradually increases an organism's sensitivity to the first event, and many of the same circuits that contribute to habituation and sensitization in *Aplysia* are involved. As another example, the learning that results from repeatedly pairing events with rewards or punishment (discussed in [Chapter 5](#)) depends on active exploration by the individual doing the learning. Understanding the relationships between different learning processes is one of the main goals of learning and memory research.

Chapter 3 Review

Know Your Key Terms

[acoustic startle reflex](#)

[cochlear implant](#)

[constraint-induced movement therapy](#)

[cortical plasticity](#)

[dishabituation](#)

[dual process theory](#)

[electrodermal activity \(EDA\)](#)

[familiarity](#)

[habituation](#)

[heterosynaptic](#)

[homosynaptic](#)

[novel object recognition task](#)

[orienting response](#)

[perceptual learning](#)

[place cell](#)

[priming](#)

[receptive field](#)

[sensitization](#)

[sensory prosthesis](#)

[spatial learning](#)

[spontaneous recovery](#)

[statistical learning](#)

[stroke](#)

[synaptic depression](#)

[word-stem completion task](#)

Quiz Yourself

1. If you are nervous about driving in the rain because you once slid off the road when it was raining, you may have become _____ to driving in the rain. ([p. 82](#))

2. If you don't mind getting fillings at the dentist because you've had so much dental work in the past, you probably are _____ to dental drills. ([p. 83](#))
3. If you've become habituated to the beeping of a fire alarm with a low battery near your bedroom, then having a crow attack you in your bedroom may _____ your responses to the beeping. ([p. 79](#))
4. Researchers can tell when a baby perceives an object as novel by monitoring the duration of its _____. ([p. 78](#))
5. When stimuli are _____ in intensity, habituation is faster. ([p. 80](#))
6. Long-term habituation is more likely to occur after _____ exposure than after _____ exposure. ([p. 81](#))
7. A physiological response that researchers sometimes use to monitor the effects of habituation and sensitization is the _____. ([p. 83](#))
8. The suggestion that sensitization and habituation are independent processes is a major feature of _____. ([p. 84](#))
9. When an organism learns but does not provide observable evidence that learning has occurred, the process is referred to as _____. ([p. 81](#))
10. The _____ task makes it possible to determine when a rat is familiar with a particular object. ([p. 87](#))
11. Sensitization of the gill-withdrawal reflex in *Aplysia* is associated with increased release of _____ by _____. ([p. 100](#))
12. Learning-related changes in the receptive fields of cortical neurons provide evidence of _____. ([p. 103](#))
13. Neurons in the hippocampus that respond strongly in particular locations are called _____. ([p. 107](#))
14. Repeated exposures to highly stressful events during development can lead to _____. ([pp. 113–114](#))

15. Devices called _____, created to facilitate sensory processing in patients who have lost some sensory function, work better over time because of _____. (p. 114)

Concept Check

1. A weight lifter repeatedly lifts a barbell. After several repetitions, he begins lifting it more infrequently, until eventually he stops. Would you say this is habituation? Why or why not?
2. A common example of sensitization is the experience of walking down a dark alleyway at night. The setting may produce feelings of nervousness, which lead to heightened arousal: you'll jump if you hear a noise behind you. Can you think of any situations (other than movies) in which people are intentionally sensitized?
3. After reading this chapter, you'll be familiar with at least some of the information it contains. If you read the chapter again, you may learn even more. Is this an example of learning from repeated events?
4. [Chapter 2](#) describes a study in which structural MRIs of London taxi drivers were compared with those of control participants who did not drive taxis ([Maguire et al., 2000](#)). In that study, researchers discovered that the size of the hippocampus in a taxi driver was correlated with the number of years the person had been driving a taxi. Why might that be?

Answers to Test Your Knowledge

Maximizing Habituation

1. The weaker the stimulus, the faster the habituation, so one option is to try to make the stimulus—in this case, the itch—less intense, perhaps by using lotion or by wearing an undershirt.
2. Sensitization is associated with increased arousal. If you decrease your arousal—say,

by going to sleep—you will decrease the likelihood of sensitization. Sleeping, or just meditating, will also make the stimulus weaker, further increasing the rate of habituation. Avoid stimulants like caffeine.

3. Assuming that you've slathered on the lotion so that the itching is not too intense, wear the shirt all the time. The more you wear it, the more your responses will become habituated to its itchiness. You might also try using fabric softener.

Perceptual Learning Versus Habituation

1. This dog show judge has learned to perceptually discriminate dogs based in part on their teeth or other features of their mouth. The dog's response has become habituated to strangers sticking their hands and face near its mouth.
2. This man has probably learned to perceive differences among wines based on odors—an example of perceptual learning. He has also presumably gotten used to wearing coats and ties.
3. Repeated experiences have caused this homeless man to be accustomed to being ignored by the general public and have likewise caused these pedestrians to be accustomed to seeing destitute people on the street.

Synaptic Mechanisms of Learning in Aplysia

1. The reduction in synaptic connections indicates habituation; less glutamate is being released in the stimulus–response pathway.
2. The increase in glutamate release indicates sensitization; stimulus–response pathways are more likely to become active.
3. The increased number of connections suggests sensitization; the increase in connections should increase the amount of glutamate released in stimulus–response pathways.
4. The number of action potentials generated in motor neurons generally reflects the amount of glutamate arriving at receptors; if this number is reduced, glutamate release

by sensory neurons is probably reduced, suggesting habituation.



Chapter 4 Classical Conditioning

Learning to Predict Significant Events



wim claes/Shutterstock

Behavioral Processes

Basic Concepts of Classical Conditioning

Refining the Basic Principles

[Error Correction and the Modulation of US Processing](#)
[Stimulus Attention and the Modulation of CS Processing](#)
[Other Determinants of Conditioning](#)

Brain Substrates

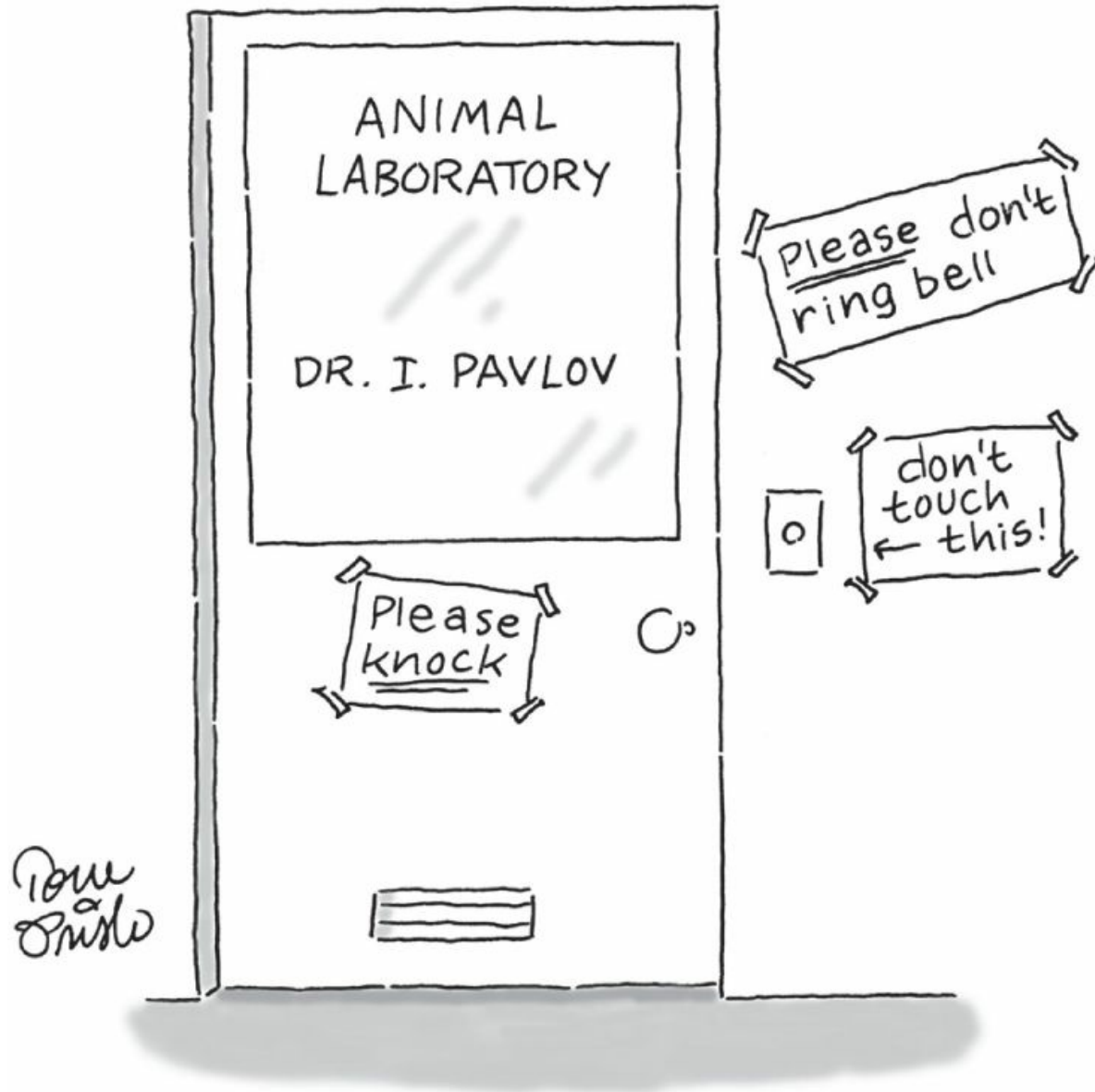
[Conditioning of Motor Reflexes in the Mammalian Brain](#)
[Invertebrates and the Cellular Basis of Learning](#)

Clinical Perspectives

[Tolerance to Addictive Drugs](#)
[*Learning and Memory in Everyday Life: Extinguishing a Drug Habit*](#)
[Reducing the Need for Medication or Reducing Its Side Effects](#)

WHAT DO THE FOLLOWING FOUR people have in common? Four-year-old Moira, who screams with delighted anticipation for ice cream when she hears the jingle of the vendor's truck in the distance; Dan, who starts feeling anxious about the carpeting in his home when he sees rain clouds on the horizon and realizes that he left the windows open this morning; Nathalie, a former cigarette smoker, who always has an urge to light up after sex; Sharon, who broke up with her ex-boyfriend years ago but still finds the sound of his voice arousing. It's not immediately apparent, but a little investigation will show that the link connecting Moira, Dan, Nathalie, and Sharon's reactions is Ivan Pavlov—or to be more precise, Ivan Pavlov's principle of classical conditioning. They have all had their responses to certain stimuli altered by classical (or "Pavlovian") conditioning.

Most people, even if they have never taken a psychology course, are vaguely aware of the story of how Ivan Pavlov (1849–1936) trained, or "conditioned," his dogs to salivate to cues such as bells or tones that predicted the impending delivery of food. References to "Pavlov's dogs" can be found throughout popular culture—in movies, TV shows, and many cartoons—as shown here and elsewhere in this chapter. [Chapter 1](#) briefly introduces Pavlov and his training method; this chapter explains why his work was so important and influential and why it continues to be relevant to experimental and clinical studies today.



Tom Prisk/www.CartoonStock.com

Why might a visitor ringing the doorbell interfere with Pavlov's experiments?

Like many other advances in science, Pavlov's discovery of classical conditioning was accidental. He was originally studying digestion, and he noticed that his dogs often started salivating even before they received their daily meat rations—for example, when they saw the bowl that usually contained their food or when they heard the footsteps of the laboratory assistant who fed them ([Pavlov, 1927](#)). Initially, Pavlov viewed the premature salivation as a nuisance that interfered with his efforts to understand how the digestive system responds to food.

Soon, however, Pavlov realized that he had stumbled on a way of studying how associations are formed in the brain of a dog.

Pavlov and his assistants began a systematic study of factors that influence how an animal learns. By keeping the dogs in restraints and collecting saliva through a tube surgically inserted into their mouths, Pavlov could measure salivation in response to various cues ([Figure 4.1](#)). He began one study by first training a dog that a doorbell always preceded delivery of food; over many paired doorbell–food trials, the dog developed a stronger and stronger salivation response to the sound of the doorbell. This form of learning, in which an animal learns that one stimulus (such as a doorbell) predicts an upcoming important event (such as delivery of food), is known today as [classical conditioning](#), or sometimes as [Pavlovian conditioning](#) (they mean the same thing).



classical (Pavlovian) conditioning

A form of learning in which an animal acquires the expectation that a given stimulus predicts a specific upcoming important event.

For the 1928 Broadway show *Paris*, Cole Porter wrote, “Birds do it, bees do it, even educated fleas do it, ...” Although Porter was referring to falling in love, he could just as well have been writing about classical conditioning. Humans, rabbits, rats, sea snails ... even insects such as fleas and flies can be trained using classical conditioning methods. Moreover, the neural substrates for every step of some forms of classical conditioning have been measured in precise detail, from the initial sensory input to the commands that drive the resulting motor responses. Avidly studied today by psychologists, neuroscientists, and clinical neuropsychologists, classical conditioning has implications far beyond what Pavlov could have anticipated when he made his accidental discovery almost 100 years ago.

4.1 Behavioral Processes

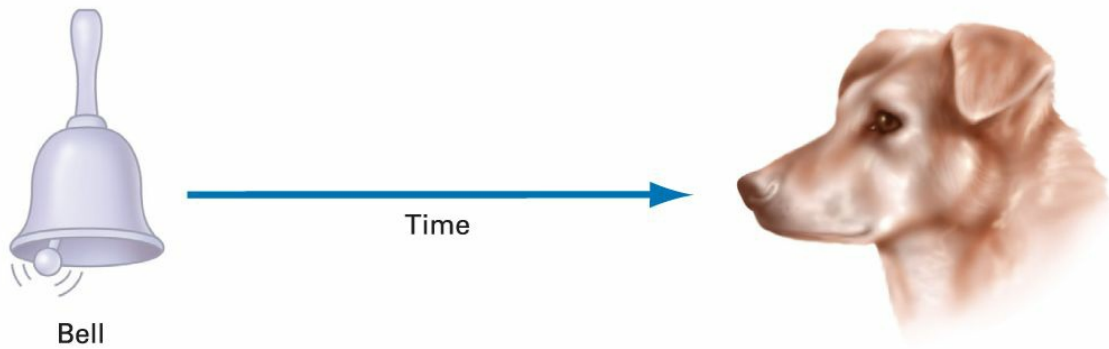
Classical Pavlovian conditioning is a way of learning about one's environment. As a child, Moira learned that the distant sound of a certain jingle predicts the imminent arrival of an ice cream truck. She loves ice cream, and she can exploit her foreknowledge of the truck's arrival by asking her mother for money now so she can be ready at the curb when the truck approaches. This is an example of learning to anticipate a desirable event so as to take maximal advantage of it. Being able to anticipate unpleasant events is also useful. If Dan is surprised by a sudden midday rainstorm, he must rush home from work to close all the windows to keep the rain from blowing in and soaking his carpets. Had he anticipated the rainstorm earlier, Dan could have closed the windows before he left for work.

It is important to note that whether or not Moira correctly anticipates the ice cream truck, it still comes at the same time. Similarly, whether or not Dan is able to predict the rainstorm, the storm still arrives. Thus, the feature that defines these examples of learning as classical conditioning is the increase in Moira and Dan's ability to predict future events, such as ice cream trucks or rainstorms. While Moira and Dan may also take some anticipatory action that makes this predictive ability beneficial to them (e.g., getting money from Mom or closing the window), their actions have no impact on whether, or when, the ice cream truck or rainstorm arrives. Classical conditioning is fundamentally about predicting future events over which we have no control, for the purpose of exploiting that foreknowledge to our benefit.

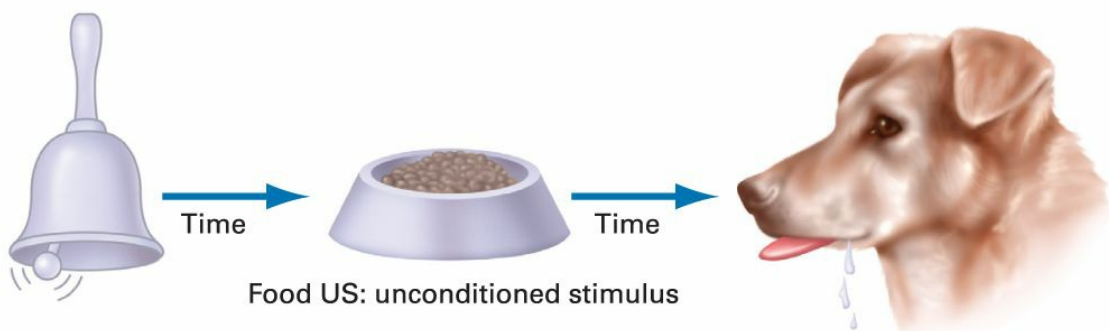
Basic Concepts of Classical Conditioning

A dog will naturally salivate when it sees or smells food. No learning is needed for it to make this response. For this reason, psychologists call the food an **unconditioned stimulus**, or **US**, which is a stimulus that naturally—that is, without learning or conditioning—evokes some response. An unconditioned stimulus, such as food, evokes a natural response, such as salivation, which psychologists call the **unconditioned response**, or **UR**; the relationship of the US and UR does not depend on learning. They both occur unconditionally without prior training. In contrast, a neutral stimulus, such as a bell that the dog has not heard before, evokes no such salivation by the dog (**Figure 4.2A**).

A Before training



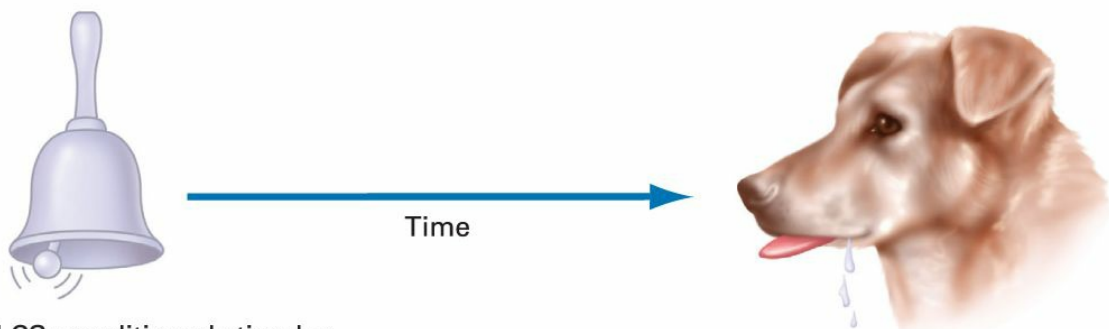
B During training



Bell CS: conditioned stimulus

Salivation UR: unconditioned response

C After training



Salivation CR: conditioned response

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FIGURE 4.2 Schematic illustration of Pavlov's experiment (A) Before training, a neutral stimulus, such as a bell, evokes no response from a dog. In contrast, the presence of food, an unconditioned stimulus (US) would naturally evoke an unconditioned response (UR), salivation. (B) During training, the bell, formerly a neutral stimulus, becomes a conditioned stimulus (CS) when it is repeatedly paired with the food US to evoke a salivation UR. (C) After training, the bell, now a conditioned stimulus (CS), evokes a learned response, the conditioned response (CR) of salivation.

Description

The first illustration is labeled Before training. Toward the left shows a bell. an arrow pointing from the bell reads,

Time and points to a dog. The dog does not respond to the sound of the bell, before training.

The second illustration is labeled During training. Toward the left shows a bell and an arrow that reads, Time. A foreground text reads, Bell C S: conditioned stimulus. It leads to a bowl of food, with a text that reads, Food U S: unconditioned stimulus. It further has an arrow that reads, Time, and points to a dog. A foreground text reads Salivation U R: unconditioned response. The dog sticks its mouth as a response to the ringing of the bell.

The third illustration is labeled After training. Toward the left shows a bell and, and an arrow that reads, Bell C S: conditioned stimulus. It leads to an arrow, that reads, Time, which further points to a dog. A foreground text reads Salivation C R: conditioned response. The dog sticks its mouth as a response to the ringing of the bell, even without the presence of the bowl.

unconditioned stimulus (US)

A cue that has some biological significance and that, in the absence of prior training, naturally evokes a response.

unconditioned response (UR)

The naturally occurring response to an unconditioned stimulus (US).

How Pavlov Conditioned Dogs to Salivate

After Pavlov put his dogs into the apparatus shown in [Figure 4.1](#), he repeatedly paired the bell with food: each time the bell was rung, an assistant promptly delivered food to the dog. This resulted in the formerly neutral stimulus, the bell, becoming a **conditioned stimulus**, or **CS**, as illustrated in [Figure 4.2B](#). After repeated presentations of the bell CS and the food US, the two became linked in the dog's mind. This training—or conditioning, as Pavlov called it—resulted in the dog learning something new: the bell predicts the food. We can evaluate the degree to which the dog learned this prediction—that is, how strongly it expects the food when it hears the bell—by measuring how much the dog salivates to the bell alone. This is shown in [Figure 4.2C](#), in which the bell alone, the conditioned stimulus (CS), now automatically evokes the same or a related response, called the **conditioned response**, or **CR**, even in the absence of the food.

conditioned stimulus (CS)

A cue that is paired with an unconditioned stimulus (US) and comes to elicit a conditioned response (CR).

conditioned response (CR)

The trained response to a conditioned stimulus (CS) in anticipation of the unconditioned stimulus (US) that the CS predicts.

For Moira, the truck jingle is a conditioned stimulus (CS), which, after being repeatedly paired with the arrival of the ice cream truck, evokes an anticipatory conditioned response (CR) consisting of Moira asking her mother for money and running to the street corner. When this response has been fully learned, Moira will be waiting at the street corner, money in hand, when the unconditioned stimulus (US)—the ice cream truck—arrives at her house. For Dan, the dark

clouds in the sky are an ominous conditioned stimulus (CS), which have become associated with subsequent heavy rains through his past experiences. These experiences have taught him to close all the windows in his house (the CR) before the arrival of the rain, the unconditioned stimulus (US).

In all three examples—Pavlov’s dog, Moira, and Dan—a learned association between a CS and subsequent US generates a CR that follows the CS. [Table 4.1](#) reviews these terms and their application in the three examples. Note that sometimes the CR is the same as the UR (as is salivation in Pavlov’s dog studies), and sometimes it is different (as with Moira and Dan). These four terms—US, UR, CS, CR—are used throughout this chapter as well as the rest of the book, so be sure you are comfortable with all of them before moving on. After reviewing [Table 4.1](#), see if you can apply what you have learned to another example in the [Test Your Knowledge](#) box.

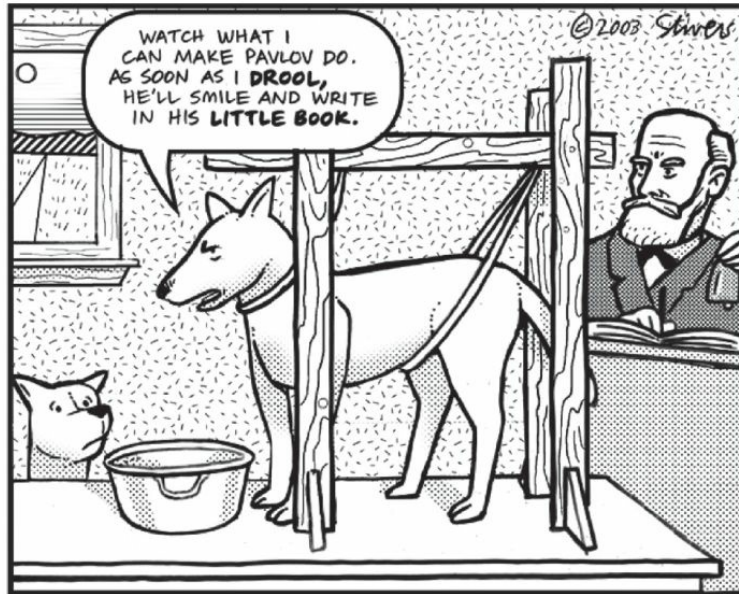
TABLE 4.1 Terminology of Pavlovian conditioning, with examples

	Unconditioned stimulus, US	Unconditioned response, UR	Conditioned stimulus, CS	Conditioned response, CR
Pavlov’s dog	Food	Salivation	Bell	Salivation
Moira	Ice cream truck	Appetite for ice cream	Truck jingle	Get money from Mom, run to corner before truck arrives
Dan	Rain	Dismay at carpet damage	Dark clouds	Closing windows before the rain starts

Test Your Knowledge

Pavlov’s Experiment

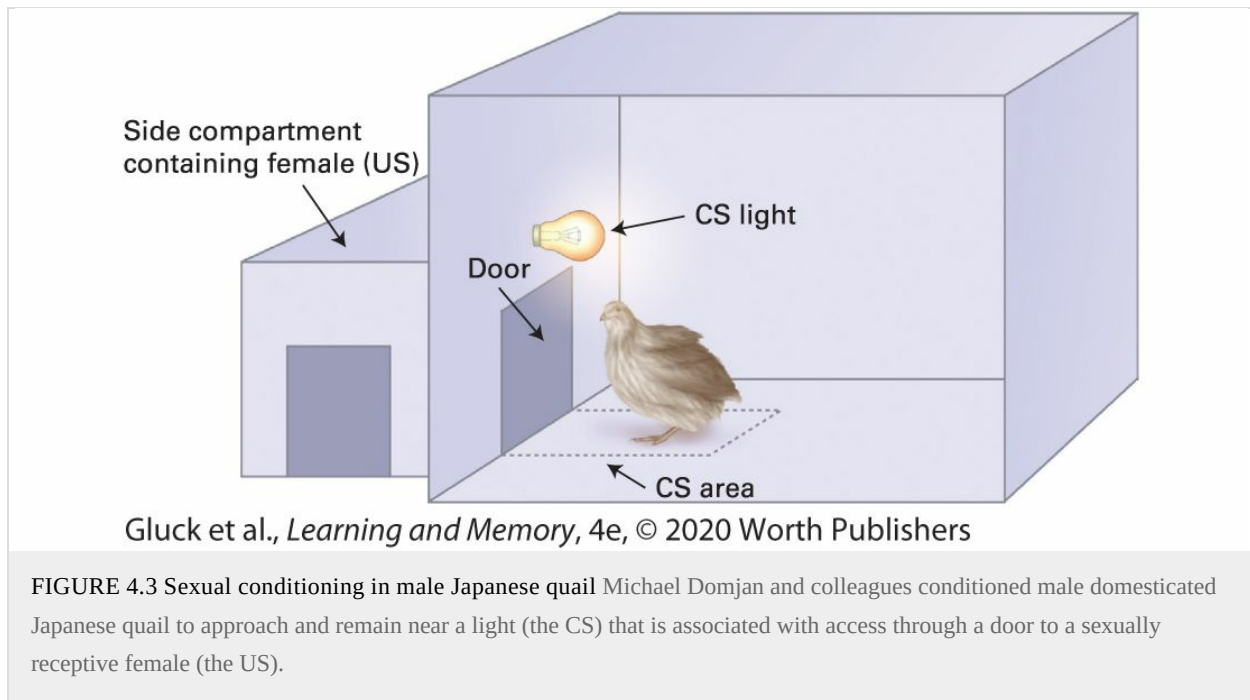
[Table 4.1](#) describes Pavlov’s experiment from the perspective of the experimenter, Pavlov. From the dog’s perspective, however, the scenario seems quite different. Using the cartoon here, identify Pavlov’s CS and CR, as viewed by the dog. ([Answers appear at the end of the chapter.](#))



Mark Stivers

Appetitive Conditioning

When the US is a desirable event (such as food delivery for Pavlov's dog or ice cream for Moira), the conditioning is called **appetitive conditioning**. In general, appetitive conditioning consists of learning to predict something that satisfies a desire or an appetite. Food and sex are among the most powerful of appetitive USs. Recall Sharon, one of the four people described at the beginning of the chapter; she was conditioned to the sound of her ex-boyfriend's voice by its past association to her having sex with him. Michael Domjan and colleagues have studied a similar form of conditioning using male domesticated Japanese quail, who will copulate readily with a sexually receptive female (**Figure 4.3**). When an arbitrary stimulus, such as a light CS, is paired repeatedly with access to a sexually receptive female (the US), the male quail exhibits a CR of approaching and remaining near the light ([Domjan, Lyons, North, & Bruell, 1986](#)).



Description

A Japanese mail quail is kept inside a huge compartment. the quail stands inside an area marked with dotted lines, labeled C S area. A rectangular area in front of the quail is labeled Door. A bulb is kept above the door, labeled C S light. Another smaller cube-like compartment is kept adjacent to the huge box. It is labeled, Side compartment containing female (U S).

appetitive conditioning

Conditioning in which the US is a desirable event (such as food delivery).

Aversive Conditioning

Dan's learning that clouds predict rain damage to his home (if the windows are not shut) is an example of **aversive conditioning**, learning to predict an expected undesirable event. Many of the procedures used for the experimental study of conditioning are examples of aversive conditioning.

aversive conditioning

Conditioning in which the US is a disagreeable event (such as a shock or an airpuff to the eye).

[Chapter 1](#) introduces both B. F. Skinner, one of the founders of the behaviorist approaches to the science of learning, and W. K. Estes, a founder of mathematical psychology and learning theory. In the early 1940s, Estes was a graduate student of Skinner's. The United States had not yet

entered World War II, but in London, England, people were already experiencing nighttime bombings by the Luftwaffe, the German air force. As Londoners heard the German bombers approaching, they stopped whatever they were doing—eating, walking, talking—and waited for the explosions. After the bombs dropped elsewhere and people realized they were safe, they resumed their interrupted activities.



Popperfoto/Getty Images

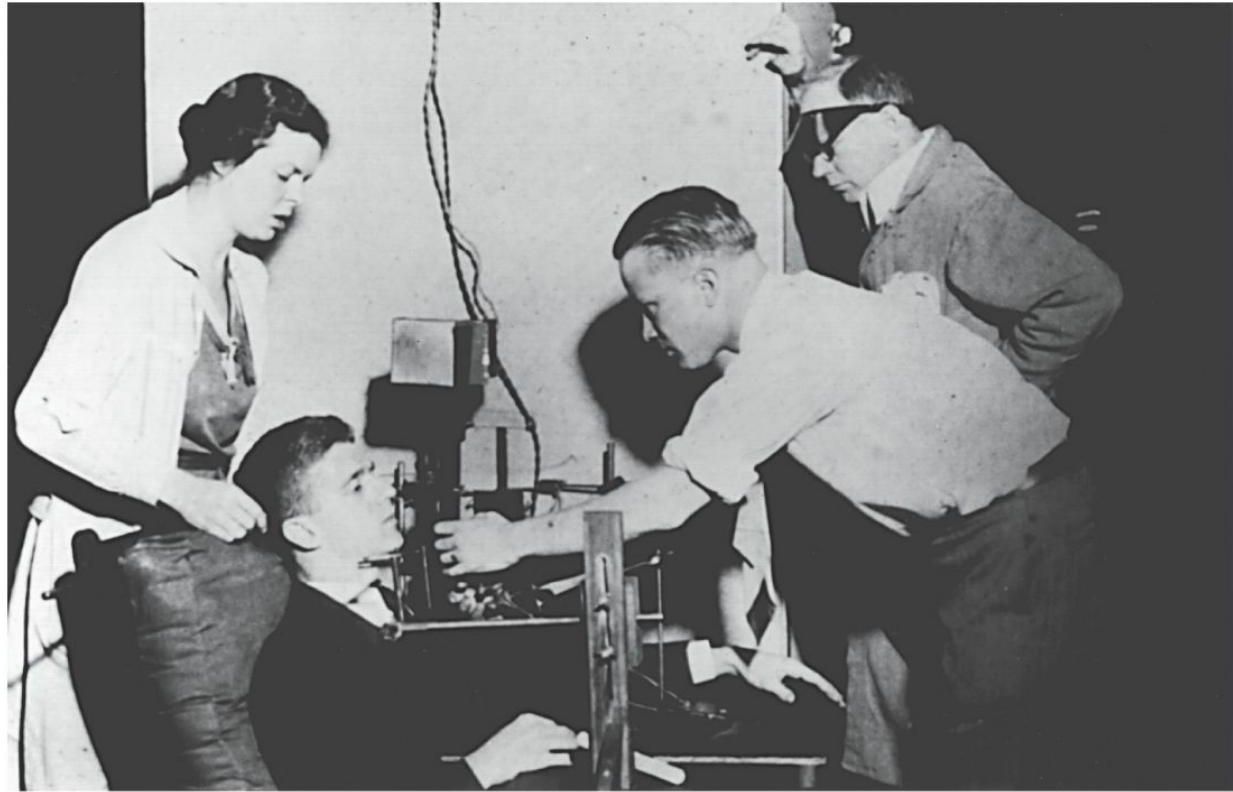
If the sound of German planes overhead is the CS that causes this family to stop eating, what is the CS that tells them it is now safe to resume their meal?

Intrigued by these stories from London, Estes and Skinner developed a new conditioning procedure for rats that was similar, in some respects, to what they imagined Londoners were experiencing. This procedure, which they called the *conditioned emotional response (CER)*, was a technique for studying learned fear ([Estes & Skinner, 1941](#)). Estes and Skinner placed hungry rats in a cage that delivered food pellets whenever the rats pressed a lever. The cage also had a metal grid floor wired to deliver a mild shock to the rats' feet. Normally, the hungry rats busily pressed the lever to obtain food, but if the experimenters trained the rats to learn that a tone (the conditioned stimulus, or CS) predicted an upcoming shock (the unconditioned stimulus, or US),

the rats would freeze (their conditioned response, CR) when they heard the tone, interrupting their lever presses and waiting for the shock. Measuring this freezing behavior allowed Estes to quantify trial-by-trial changes in the learned response. Within a few years, this conditioned emotional response procedure became one of the most widely used techniques for studying animal conditioning, and it is still in use today. ([Chapter 10](#) describes further details of fear conditioning and how emotions, such as fear, influence learning and memory.)

Mammalian Conditioning of Motor Reflexes: Eyeblink Conditioning

Another widely studied form of aversive conditioning—one that induces an anticipatory defensive response much like Dan’s shutting his windows—is [eyeblink conditioning](#), perhaps the most thoroughly studied form of motor reflex conditioning in mammals ([Gormezano, Kehoe, & Marshall, 1983](#)). [Chapter 1](#) discusses Clark Hull, one of the early mathematical psychologists; he used (and perhaps abused) his graduate students by teaching them to blink in anticipation of a slap to the face, as shown in [Figure 4.4](#). The participant in this photo is Ernest “Jack” Hilgard, who later went on to become a pioneer in the study of the psychology of hypnosis and the author of a leading textbook on memory.



Courtesy of Elizabeth Jecker

FIGURE 4.4 Face-slap eyeblink conditioning in the 1920s Clark Hull (standing with visor) and his young graduate student Ernest Hilgard (seated) in an early study of classical eyeblink conditioning at Yale University. Hull trained Hilgard to blink in anticipation of a slap to the face.

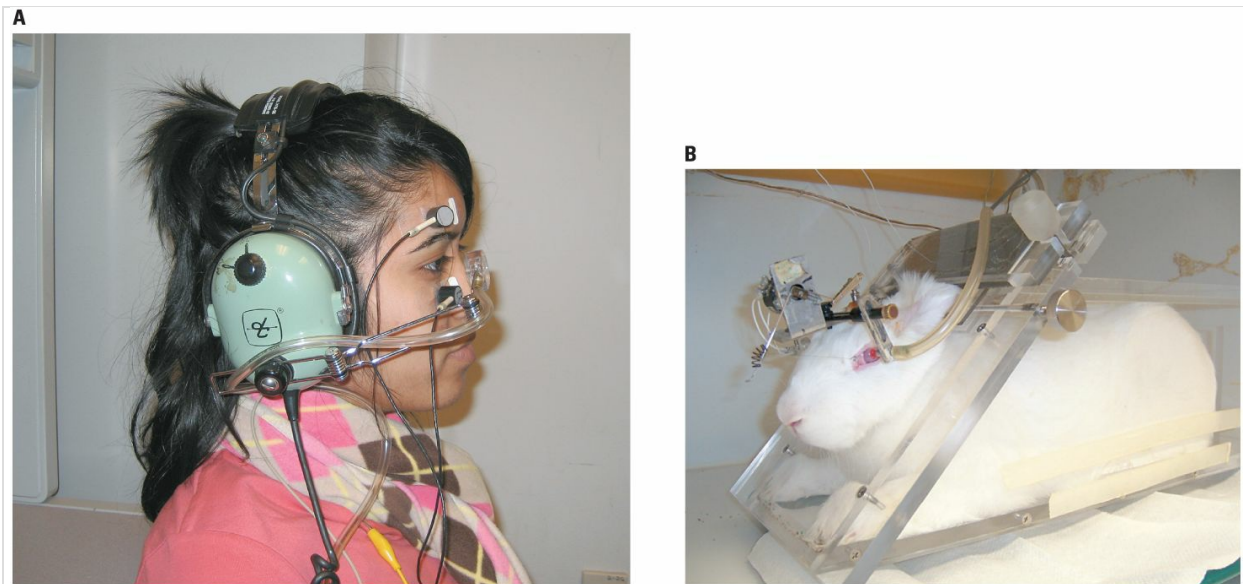
eyeblink conditioning

A classical conditioning procedure in which the US is an airpuff to the eye and the conditioned and unconditioned responses are eyeblinks.

Hull arranged for a tone (CS) to play just prior to each face slap (US). After many repeated presentations of the CS followed by the US, poor Jack began to blink (the CR) every time he heard a tone, whether or not it was followed by a slap.

For practical as well as ethical reasons, researchers no longer use the face slap as a US in human eyeblink conditioning. Instead, they often use an airpuff to the eye. This is not painful, but it does cause a reflexive—that is, unconditioned—eyeblink UR. (If you don't believe this, ask a friend to blow lightly in your eye.) The blink UR in human eyeblink conditioning can be measured in several different ways, one of which is through the use of electromyography (EMG) detectors of electrical activity of muscles, placed above and below the eye, as shown in [Figure](#)

4.5A. The CS is a tone, typically delivered through headphones so that the volume can be regulated (and outside noises masked). With repeated pairings of the tone CS and airpuff US, subjects develop a CR: in this case, an anticipatory blink that occurs before US arrival, such that the eye is partially shut and partially protected when the airpuff occurs.



A: Mark Gluck; B: Richard F. Thompson

FIGURE 4.5 Eyeblink conditioning in humans and rabbits (A) In human eyblink conditioning, a tone CS is delivered through headphones. The US is a puff of air delivered through the rubber tube. The eyblink CR is recorded by EMG electrodes placed above and below the eye. (B) In rabbit eyblink conditioning, a similar rubber tube delivers the airpuff US to the rabbit in the restraining acrylic glass case; a photobeam measures the CR and UR.

Description

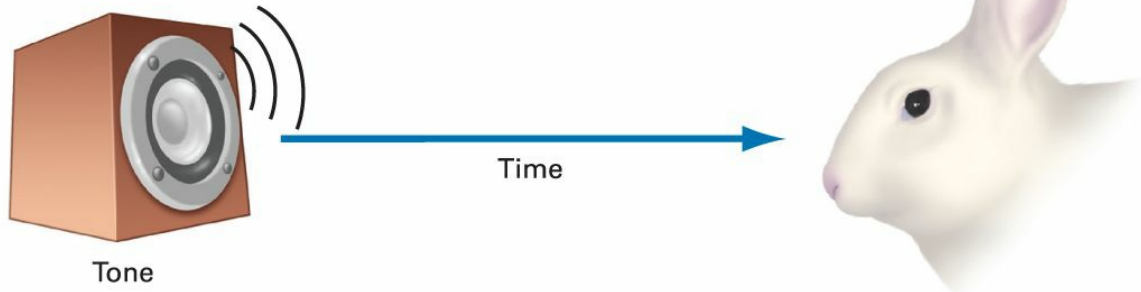
The photo on the left shows a young girl with her face wrapped with earphones and rubber tubes. she is a part of experimenting eyblink condition in humans. The photo on the right shows a rabbit, with its face wrapped with earphones, rubber tubes, and acrylic glass as a part of eyblink experiment.

What is most important about eyblink conditioning is the similarity of its appearance in many different species, so that the results found in one species can reasonably be expected to apply to others. Eyblink conditioning has been shown in mice, rats, and monkeys, but one of the most common animals for the study of eyblink conditioning has been the rabbit because of its propensity to sit still for long periods and to blink very infrequently except when something bothers its eyes. [Figure 4.5B](#) shows a rabbit in a restraining acrylic glass case within which eyblink conditioning is often conducted. The tube at the rabbit's left eye delivers the airpuff US,

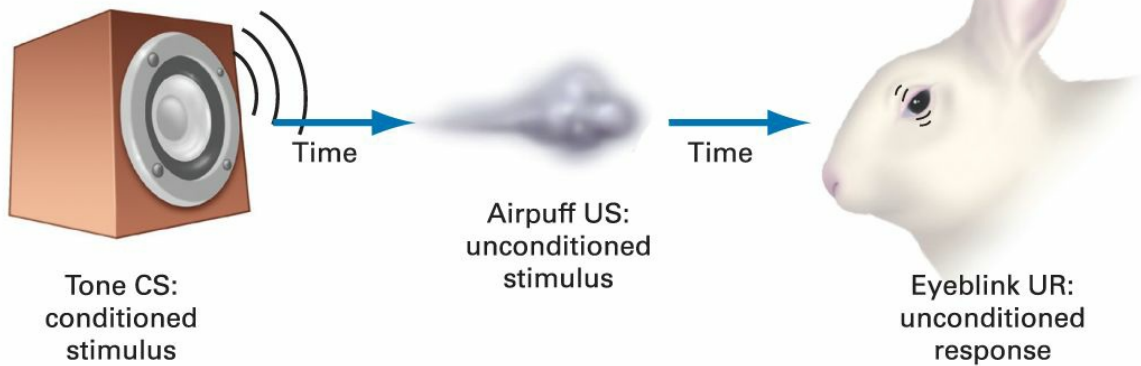
while the eyeblink CR and UR are measured by photo beams.

With rabbits, just as with humans, the airpuff is the US, and the reflexive blink is the UR, as shown in [Figure 4.6](#). Before training, the tone does not cause the rabbit to blink because it is a neutral stimulus, as shown in [Figure 4.6A](#). However, if the airpuff US is repeatedly preceded by a tone, as shown in [Figure 4.6B](#), the animal learns that the tone predicts the airpuff US and is a warning signal to get ready. Eventually, the animal will blink as a response to the tone alone, as shown in [Figure 4.6C](#). At this point, the tone has become a CS, and the anticipatory eyeblink is the CR.

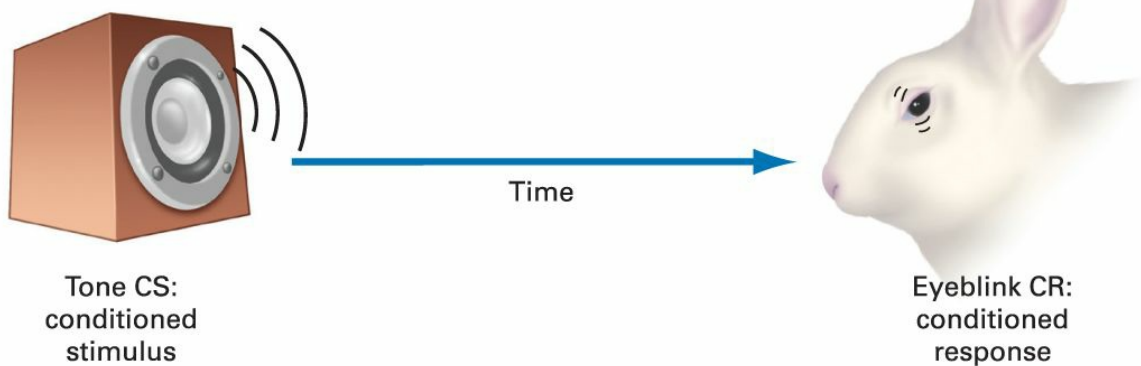
A Before training



B During training



C After training



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FIGURE 4.6 Learning progression in rabbit eyeblink conditioning (A) Before training, the tone is a neutral stimulus that has no relevance to the rabbit. (B) The tone CS followed by airpuff US causes an eyeblink UR in a naive rabbit. (C) The tone CS is followed by a blink CR in a rabbit that has undergone eyeblink conditioning. Compare these diagrams to [Figure 4.2](#), which shows analogous stages of Pavlov's conditioning procedure.

Description

The first illustration is labeled Before training. Toward the left is a music instrumental box, which is labeled Tone. An arrow steering from the music box reads, Time and it points to a rabbit on the right. The rabbit does not show any indifference.

The second illustration is labeled During Training. Toward the left is a music instrumental box, which is labeled Tone C S: conditioned stimulus. It has an arrow that reads, Time, which points to an air puff. The text in the foreground reads, Airpuff U S: unconditioned stimulus. An arrow point from the air puff reads, Time. It points to a rabbit. it has an eye blink. The text in the foreground reads Eyeblink U R: unconditioned response.

The third illustration is labeled After training. Toward the left is a music instrumental box, which is labeled Tone C S: conditioned stimulus. AN arrow steering from it reads, Time and it points to a rabbit, which has its eye blinking. The text in the foreground reads, Eyeblink C R: conditioned response. The rabbit blinks its eye with the air puff after its training.

To the uninformed observer, the learned conditioned response, the eyeblink CR, is identical to the automatic unconditioned response, the eyeblink UR. However, the learned CR takes place during the warning period provided by the CS, *in advance of* the US and UR, adaptively protecting the eye from the onset of the airpuff. (Think of the CS as being analogous to a weather report predicting the onset of rain.) Similarly, in Pavlov's original salivation study, the learned CR, salivation, is the same as the dog's natural unconditioned response to food, but it takes place *before* the food is presented, at the sound of the doorbell that predicts the food.

You have now been introduced to several different formats that are used for experiments in classical conditioning: two appetitive preparations and two aversive preparations. These are all reviewed in [Table 4.2](#).

TABLE 4.2 Widely used classical conditioning procedures

	Unconditioned stimulus, US	Appetitive conditioning		
		Unconditioned response, UR	Conditioned stimulus, CS	Conditioned response, CR
Pavlov's dog	Food	Salivation	Bell	Salivation
Quail sex	Sexually available female	Approach, mounting, and copulation	Light	Approach
Aversive conditioning				
Conditioned emotional response	Shock	Freezing	Tone	Freezing
Eyeblink conditioning	Airpuff	Blink	Tone	Blink

Classical Conditioning in Everyday Life

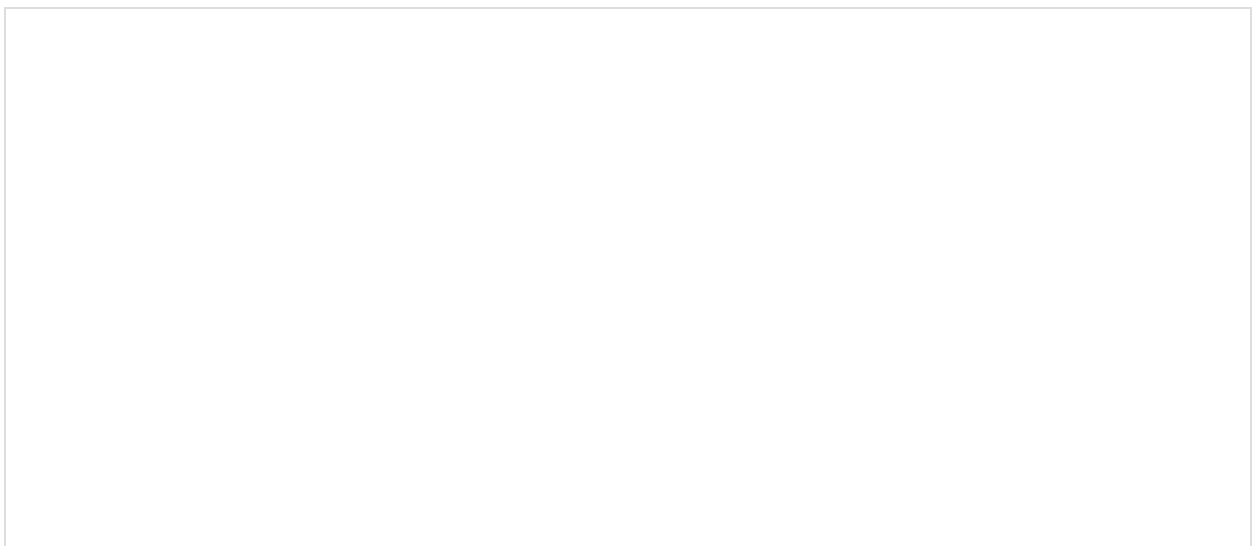
Are you sure you can tell the US, UR, CS, and CR apart? Test yourself by identifying each of them in the real-world situations described below. Also indicate whether each scenario is an example of appetitive or aversive conditioning.

([Answers appear at the end of the chapter.](#))

1. Advertisements for a new sports car show a sexy female model draped over the car's hood.
2. Mark loves pizza. When he was a boy, his parents frequently had it delivered to their home. Because the pizzas often arrived only lukewarm, his parents would put the pizza, still inside the box, into the oven to heat up. This caused the box to give off a smell of burning cardboard. Now, years later, whenever Mark smells cardboard burning, he gets hungry for pizza.

Learning a New Association

What does learning typically look like in eyeblink conditioning or other classical conditioning procedures? [Figure 4.7](#) shows an eyeblink CR becoming stronger over several days of training in a rabbit eyeblink-conditioning study. Each day, the animal received 80 training trials, each of which presented a tone followed shortly thereafter by an airpuff to the eye. The graphs (dark blue lines) in [Figure 4.7](#) show the extent to which the rabbit's eyelid lowered at the start of different days during the experiment; the higher the curve, the farther the eyelid had shut. Note that on the beginning of day 1, the only response was the eyeblink UR that occurred *after* the onset of the airpuff US. However, with training, an eyeblink CR emerged: by day 3, there was movement of the eyelid before the US arrived. This anticipatory blink in response to the CS was the beginning of a CR. With further training, by about day 5, a strong anticipatory eyeblink CR occurred, timed so that the eyelid was safely closed before the airpuff US occurred.



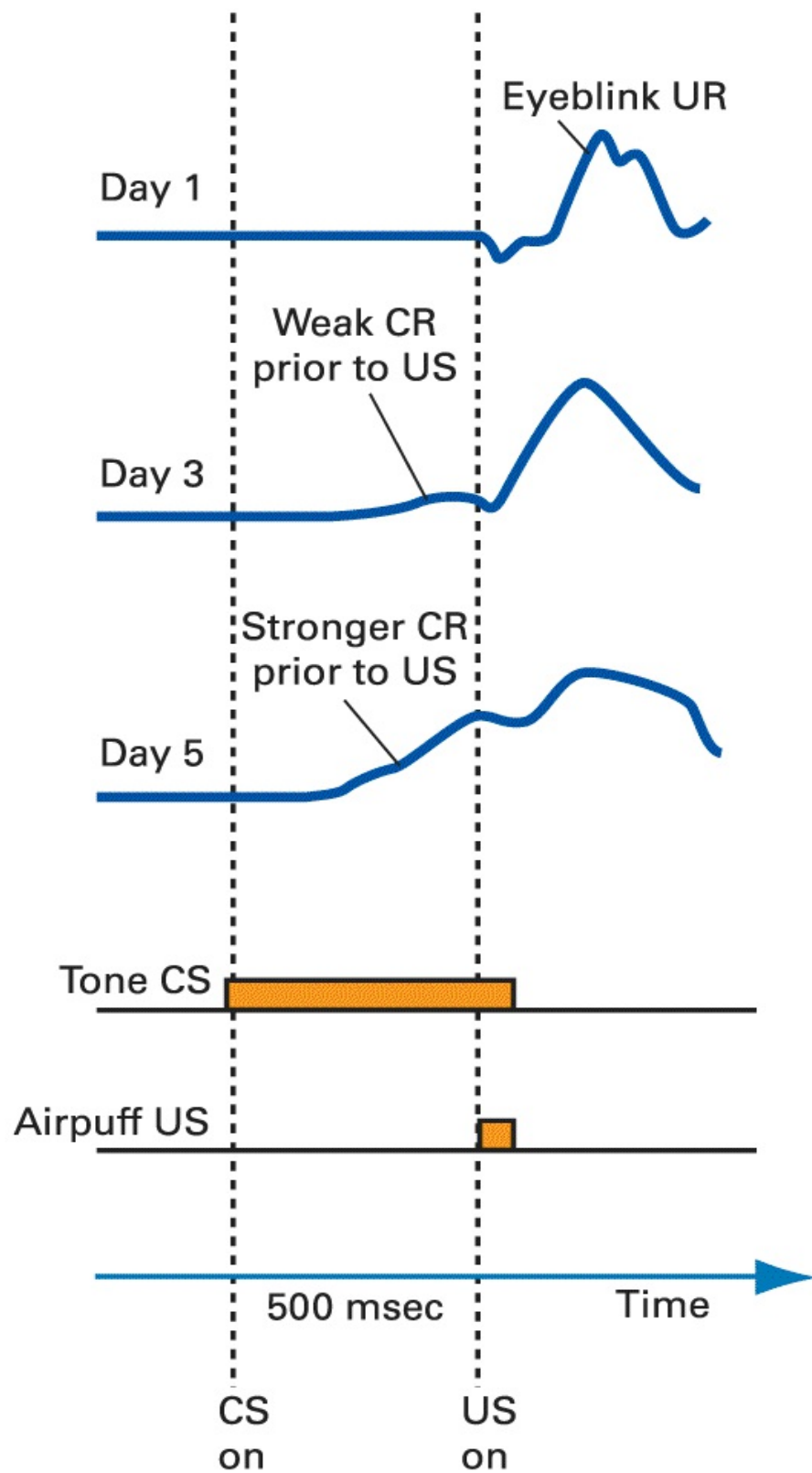
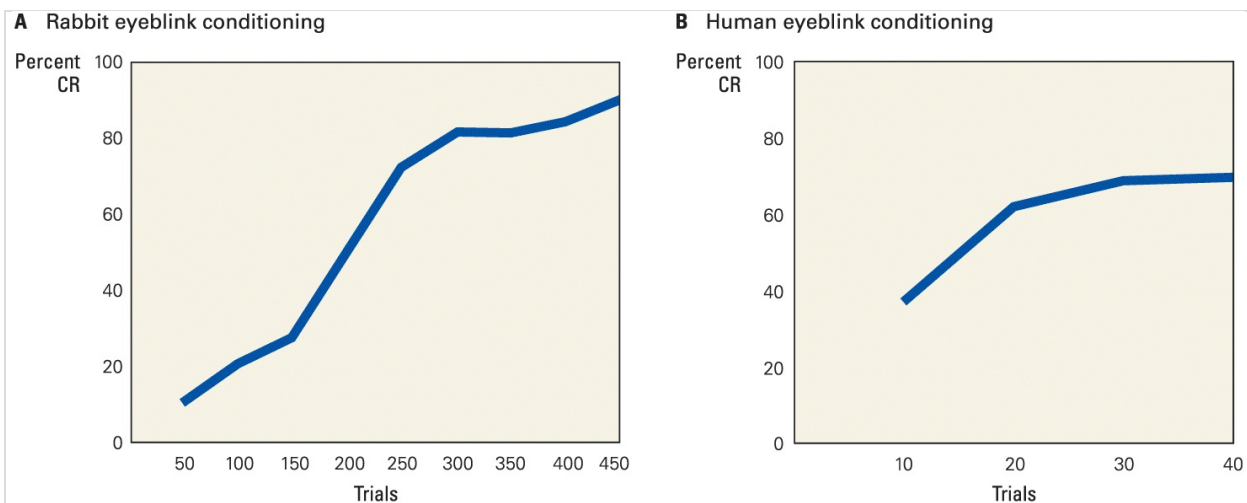


FIGURE 4.7 Acquisition of eyeblink-conditioning response Development of a conditioned response as measured at the beginning of day 1, day 3, and day 5 of training, using a standard tone–airpuff trial sequence. On day 1, only a UR to the airpuff is observed, but by day 3, an anticipatory eyeblink starts to emerge. By day 5, this anticipatory CR is strong and occurs reliably before the airpuff US.

Description

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In both rabbits and humans, eyeblink conditioning is a gradual process that occurs over many trials. [Figure 4.8](#) shows trial-by-trial changes in the percentage of human participants and rabbits giving conditioned eyeblink responses in a study of tone–airpuff conditioning in both species. Although the graphs in the figure are not identical (humans learn faster), they are quite similar. Most important to note is that in both humans and rabbits, the percentage rises over time, until most trials elicit an appropriately timed predictive eyeblink CR.



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FIGURE 4.8 Learning curves for rabbit and human eyeblink conditioning (A) A learning curve showing the percentage of CRs in rabbits across blocks of training trials. (B) Analogous learning curve for human eyeblink conditioning. Although these curves are qualitatively similar, they reflect different training regimes, since the rabbits are usually trained in blocks of 1-hour trial sessions on successive days, whereas humans are trained in a single hour-long session.

(A) Data from [Allen, Chelius, & Gluck, 2002](#); (B) Data from [Allen, Padilla, Myers, & Gluck, 2002](#).

Description

The graph on the left shows a learning curve plots the eyeblink condition in rabbits. The X-axis shows Percent C R and Y-axis shows Trials. It shows a steep learning curve until about 300 trials when C R reaches 90%.

The graph on the right shows a learning curve plots the eyeblink condition in humans. The X-axis shows Percent C R and Y-axis shows Trials. The humans show a steep learning curve from 10 to 20 trials, when C R reaches 80%

Refining the Basic Principles

Studies of classical conditioning have led to a broad range of insights into the subtle factors that influence animal and human learning. In this section, we explore four of these refinements, looking more closely at variations in the USs that develop; the constraints on what makes a cue a CS or a US; how old associations are extinguished when no longer applicable; and what happens when two cues, rather than one, are presented during learning.

Conditioned Compensatory Responses

Remember Dan, who learned (from past bad experiences) to notice dark clouds in the sky and expect rain? How do we know he is expecting the rain? Well, we might observe him close his windows. Dan also has a swimming pool out back. If he were expecting heavy rains for several days, he might worry about the pool overflowing and damaging his lawn and house. Given the weather forecast, he might think it prudent to partially drain his pool, lowering the level a few inches before the rain arrives. When the rain does come, the water level will return to the same point as before Dan drained it, reducing the likelihood of an overflow. If rain is coming, then it will come, whether or not Dan is able to predict it (so his efforts to close the window or drain the pool do not affect the weather). However, Dan's preparatory response (preemptively lowering the water level in the pool) based on his expectation of rain compensates for the expected rise in water level and ensures that the pool never gets too full.

An analogous conditioned compensatory response was demonstrated by two of Pavlov's colleagues more than 80 years ago ([Subkov & Zilov, 1937](#)). These researchers on several occasions injected dogs with adrenaline (also known as epinephrine), a chemical normally produced by the adrenal glands in response to stress or anxiety. The usual effect of adrenaline is an increase in heart rate. However, the dogs' heart rate increased less and less with each subsequent injection. A decrease in reaction to a drug, so that larger doses are required to achieve the original effect, is known as [tolerance](#). What causes tolerance to develop?

tolerance

A decrease in reaction to a drug such that larger doses are required to achieve the same effect.

To explore this question, the researchers placed their dogs on injection stands, where the dogs normally received the drug injection, but they administered a neutral, inert substance rather than the adrenaline. The researchers observed that this caused the dogs' heart rate to *decrease*. Apparently, the various cues (the stand, the injection) that predicted the adrenaline injection triggered a conditioned compensatory response that lowered the dogs' heart rate in anticipation of the adrenaline's causing an increase in heart rate. Such automatic compensatory responses occur primarily in body systems that have a mechanism for [homeostasis](#), a tendency of the body (including the brain) to gravitate toward a state of equilibrium or balance.

homeostasis

The tendency of the body (including the brain) to gravitate toward a state of equilibrium or balance.

Much like Dan the homeowner, whose expectation of a storm and rain is apparent from his reducing the level in the pool to compensate for the expected rainfall, the dogs in these studies automatically used advance information about the forthcoming adrenaline injection to compensate for the drug's effect. The automatic, learned anticipatory decrease in heart rate combined with the increase produced by the drug resulted in a lower total increase in heart rate than was experienced on the first (unexpected) administration of adrenaline. Since the dogs had been conditioned to expect adrenaline after seeing cues such as the stand or the syringe, their bodies compensated by lowering their heart rates to maintain a constant level. The same compensatory mechanisms at work in certain aspects of drug addiction are discussed in [Section 4.3](#).

What Cues Can Be CSs or USs?

The USs in a conditioning experiment are by definition events that are biologically significant, either because they are inherently pleasurable (such as food or sex) or because they are inherently disagreeable (such as shock or an airpuff to the eye). In contrast, a CS can be any cue in the environment, even a US. Thus, an airpuff to the eye—which is a US in the eyeblink-conditioning paradigm—can serve as the CS in another experiment, where, for example, an animal might learn that an airpuff predicts food delivery (the new US). Thus, stimulus cues are not inherently CSs or USs; rather, those terms define the roles the cues play in a particular learning situation.

Recall the description of Nathalie at the beginning of this chapter. She is a former smoker who

gets an urge for a cigarette after sex. In Nathalie’s case, sex is the CS that has become associated with cigarette smoking, which is the US, as shown in [Table 4.3](#). After a person gets into the regular habit of having a cigarette after sex, the craving for and expectation of cigarettes becomes the CR. (You’ll read more about addiction and conditioning later on in this chapter.) In contrast, for Sharon, who becomes aroused at the sound of her ex-boyfriend’s voice, his voice is now the CS, and her sexual arousal is her CR. Thus, for Nathalie, sex can be a CS that predicts cigarette smoking, while for Sharon, sex is the US that previously followed hearing her boyfriend’s voice. It all depends on the individual’s unique experiences.

TABLE 4.3 The Same Cue Can Be a CS and a US

	Unconditioned stimulus, US	Unconditioned response, UR	Conditioned stimulus, CS	Conditioned response, CR
Nathalie	Smoking a cigarette	Reduced craving for cigarette	Sex	Desire for a cigarette
Sharon	Sex	Sexual gratification	Ex-boyfriend’s voice	Sexual arousal

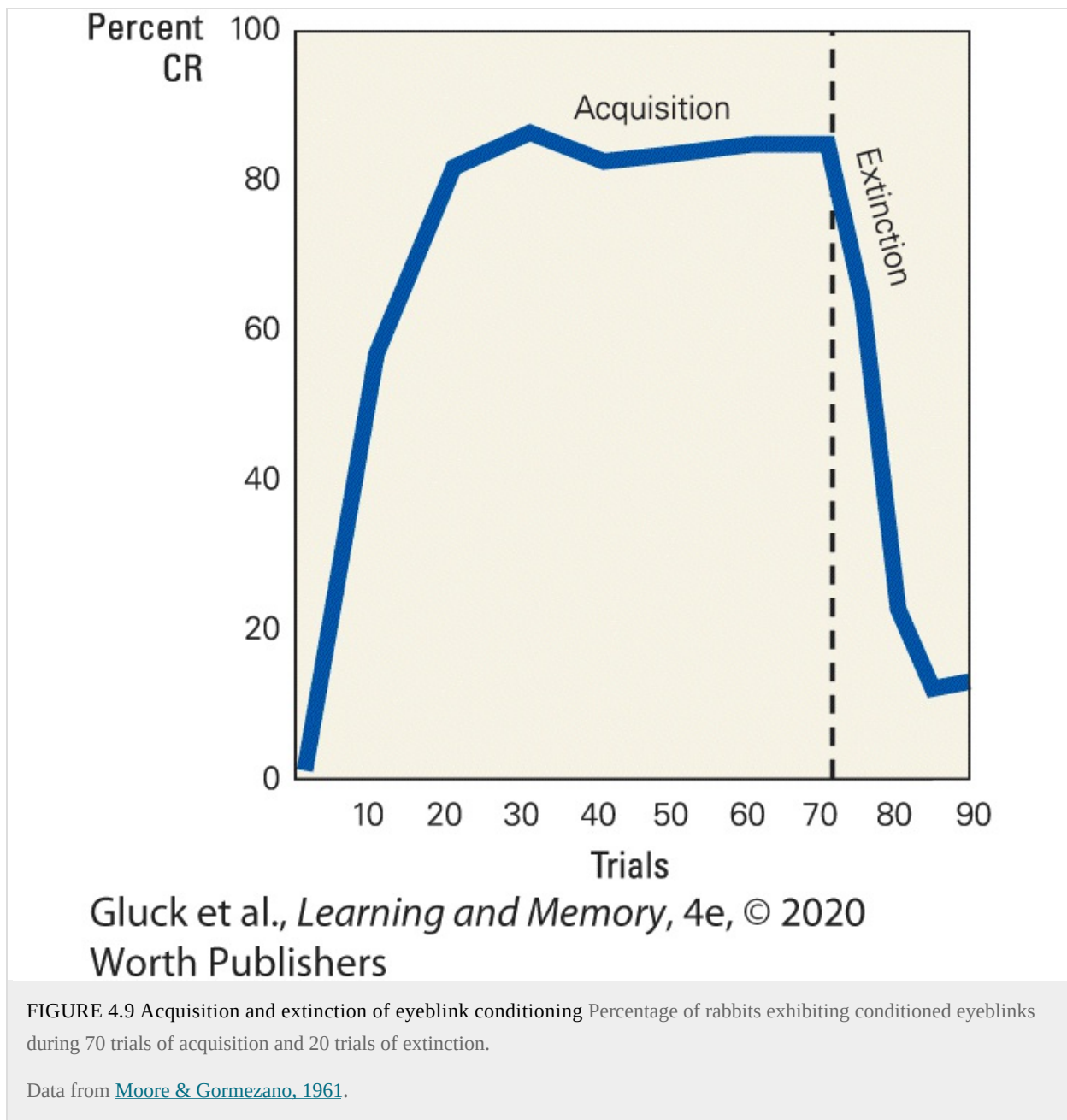
Extinguishing an Old Association

What do you think would happen if Moira moved to a new neighborhood where a neighbor’s wind chime sounded just like the jingle of an ice cream truck? Now, each time she hears the wind chime jingle, there is no ice cream truck coming, so we can expect that her excited reaction to the jingle will eventually disappear. In this case, a previously acquired association becomes diminished through repeated presentation of the CS (jingle) in the absence of the US (ice cream)—a process known as [extinction](#). It was first described in the early studies of [Pavlov \(1927\)](#), as noted in [Chapter 1](#).

extinction

In classical conditioning, the process of reducing a learned response to a stimulus by ceasing to pair that stimulus with another, previously associated stimulus.

Once eyeblink conditioning is acquired, it too can undergo extinction if the former CS (tone) is presented repeatedly without an airpuff. Eventually, the rabbit (or person), formerly conditioned to blink to the tone, begins to learn that the world has changed, and the tone no longer predicts the US. [Figure 4.9](#) shows what happens if, after 70 trials of eyeblink-conditioning acquisition training, rabbits are given 20 trials of tone-alone extinction training ([Moore & Gormezano, 1961](#)).



Description

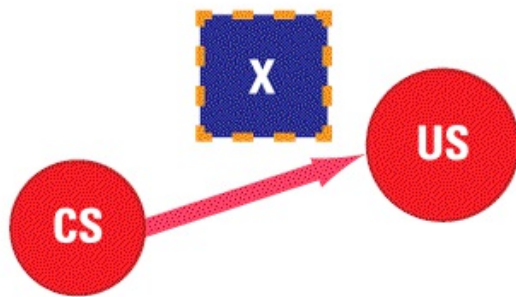
The X-axis shows Percent C R and Y-axis shows Trials. It shows the eyeblink condition during 70 trials of acquisition and 20 trials of extinction. A steep learning curve rises to almost 90% in 30 trials, and then plateaus in the 80 percents. A dotted line marks 70 trials, where the transition from acquisition to extinction begins. Later, the curve falls off rapidly to about 10% at 85 total trials.

It is tempting to think of the extinction in [Figure 4.9](#) as simply the “unlearning” (or forgetting) of the CS–US association. But that is not the case; extinction is not just unlearning. Rather, it is a

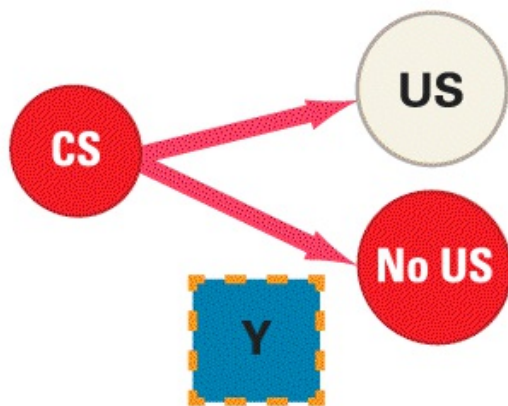
combination of unlearning and the learning of a new, opposing response to the CS. Specifically, it appears that during extinction, the CS acquires a second, “don’t respond” meaning that competes with the originally acquired “do respond” association. This suggests that even though the animal (or person) is no longer responding to the CS at the end of extinction training (as seen in [Figure 4.9](#)), the learned response is not gone; it is just suppressed or unexpressed.

Some of the most compelling evidence for this view of extinction comes from studies showing that the original learned response can reappear if the extinction is accompanied by a change in context (such as moving to another room or testing chamber). In one example, a CS is paired with a shock in one context (context X) and then extinguished in another context (context Y); then the CS is again presented in context X, and the response is renewed ([Bouton & King, 1983](#)). As noted earlier, the most parsimonious explanation seems to be that there are two associations: CS–US and CS–no US; the context determines which response is retrieved ([Bouton, 1991](#)). During initial training, a CS–US association is created in context X ([Figure 4.10A](#)); then, in context Y, a CS–no US association is created. At this point, the CS–US association learned in X has not disappeared, even though there is no responding to the CS in context Y ([Figure 4.10B](#)). When the animal is returned to context X, the original CS–US association is retrieved, and the response “spontaneously” reappears ([Figure 4.10C](#)).

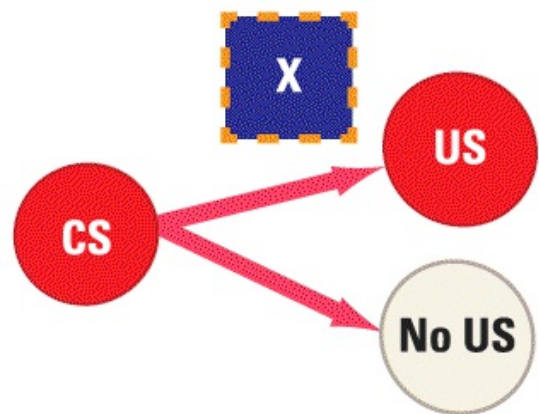
A CS–US learning in context X



B Extinction in context Y



C Renewal in context X



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

FIGURE 4.10 An account of extinction and renewal that involves context

Information from [Bouton, 1991](#).

Description

The first illustration labeled A shows C S–U S learning in context X. C S and U S are both represented in circles. An arrow, labeled X, points uphill from C S to U S.

The second illustration labeled B shows Extinction in context Y. C S, U S, and No U S are represented in circles. An arrow points from C S to U S and an arrow labeled, Y points from C S to No U S.

The third illustration labeled C shows renewal in context X. C S, U S, and No U S are represented in circles. An arrow points from C S to No U S and an arrow labeled X, points from C S to U S.

Another way in which extinction can be undone is if a long time passes before the animal is retested with a presentation of the CS. The return of a CR after such a delay is another example of spontaneous recovery (a topic discussed in [Chapter 3](#)). Spontaneous recovery is another phenomenon reported first by [Pavlov \(1927\)](#). It is seen in almost all forms of classical conditioning, which suggests that associations that become dormant following extinction training are not entirely lost.

BELL RINGS, I GET A TREAT...
BELL RINGS, I GET A TREAT... IT
WENT ON THAT WAY FOR DAYS. THEN,
OUT OF THE BLUE... BELL RINGS, I
GET **NOTHING AT ALL!!** NADA!
I MEAN, CAN YOU SERIOUSLY CALL
MY ATTACK UNPROVOKED?

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THE DARK TRUTH ABOUT PAVLOV'S DOG.

Mark Parisi/Atlantic Feature Syndicate

Identify the conditioning paradigm illustrated here.

Further evidence that extinction does not involve the total loss of what was previously learned comes from studies of rapid reacquisition, where a previously extinguished CS is learned more rapidly than a novel CS. Rapid reacquisition suggests that the learned association between a CS and a US is saved during extinction even if the behavioral CR is no longer evident following extinction trials.

Compound Conditioning and Overshadowing

When Neha was a young mother, her two children, Geeta and Sunil, would often talk to her at the same time, competing for their mother's attention. As you can imagine, it was hard for Neha to pay full attention to both kids. The same competition can occur when two cues, such as a tone and a light, appear together in a conditioning experiment—a paradigm known as **compound conditioning**. When an animal or a person is trained in a tone + light → US compound conditioning paradigm, learning of the compound stimulus usually proceeds much like learning of a simple single-cue stimulus. However, when one of the stimuli, such as the tone, is later tested alone, it becomes apparent that something different has happened. Many studies have shown that the tone will have less association with the US if it is trained as part of a compound than if it had been trained alone in a tone → US procedure. This suggests that two cues (like the two young children, Geeta and Sunil) are competing against each other, and neither produces as much learning as it would have if it had been used alone during training.

compound conditioning

Conditioning in which two or more cues are present together, usually simultaneously, forming a compound CS.

However, imagine if Geeta had a soft, quiet voice but Sunil was prone to shouting out loud. As much as their mother might want to listen to both equally, it is likely that Sunil would be the one more easily heard. This is analogous to what is seen in compound conditioning studies when one of the stimuli is much stronger than the other. If the light, for example, is very dim and hard to see but the tone is loud and salient, the tone is said to *overshadow* the light during learning. In classical conditioning, **overshadowing** occurs when a more salient cue within a compound acquires a far greater share of attention and learning than the less salient cue. For example, the learning that accrues to a loud tone in a loud tone + dim light → US compound training paradigm will be far more than the learning associated with the dim light because the loud tone is far more salient than the dim light. In contrast, if the tone were very soft and the light very bright, the opposite pattern would be seen, with the bright light overshadowing the soft tone and

accruing more learning. Even so, both cues, when tested individually, command less attention than they would have if they were used alone during training rather than in a compound.

overshadowing

An effect seen in compound conditioning when a more salient cue within a compound acquires more association strength than does the less salient cue and is thus more strongly associated with the US.

Error Correction and the Modulation of US Processing

[Chapter 1](#) introduces Aristotle’s argument that contiguity—or closeness in time and space—is necessary for learning a new association, such as that between a CS and a US. During most of the first half of the twentieth century, psychologists believed that contiguity is both necessary and sufficient: as long as a potential CS and a US occurred with little separation in time and space, animals and people were expected to form an association between the two ([Hull, 1943](#)). But would it really make sense for animals or people to learn associations between all the simultaneously occurring stimuli they perceive? Would it even be possible?

The Informational Value of Cues

Imagine you are a struggling stock investor whose livelihood depends on correctly predicting whether the stock market will go up or down the next day. One morning, Doris, an eager new stock analyst, walks into your office and says that if you hire her, she will tell you each day which way the next day’s market will go. You agree, and during her first week of work, you are amazed to see that she is 100% accurate, correctly predicting each day whether the market will rise or fall. The next week, Herman comes to visit and offers you his services as a stock analyst to predict the market’s movements. Would you hire him? Probably not, even though he is known to be as good as Doris, because he is redundant if you already have Doris; that is, Herman offers no value beyond what you are already getting from Doris. You might say that Doris’s early success at predicting the stock market has blocked you from valuing Herman’s similar, but redundant, ability to do the same, as summarized in [Table 4.4](#).

TABLE 4.4 Informational redundancy blocks learning

Group	Phase 1	Phase 2	Phase 3 (test)
Stock prediction	Doris → correct prediction	Doris & Herman → correct prediction	Hire Herman? “No way; don’t need him.”

Medical diagnosis	Patient eats peanuts → stomachache	Patient eats peanuts & licorice → stomachache	Could the licorice be causing the stomachache? “Unlikely; patient should enjoy licorice but avoid peanuts.”
Bower & Trabasso (1964). See p. 143 and Figure 4.12	Circle → A	Circle + top dot → A	Rectangle + top dot → ?
	Triangle → B	Triangle + bottom dot → B	Rectangle + bottom dot → ?
			“Participants have no idea which is A or B.”

A similar situation can occur in medical diagnosis. Consider a young physician treating a patient who has come into the emergency room on several occasions during the month of January, each time with a bad stomachache. The physician reviews what the patient ate in the day before each outbreak and finds that he had eaten peanuts before each stomachache. The physician suspects that the stomachaches are being caused by a bad reaction to peanuts. The next month, the same patient comes back to the emergency room, once again with a stomachache. Again, the physician asks him to list everything he ate the day of the outbreak. This time, the physician notices that the patient ate both peanuts and red licorice just prior to his stomachache. Although it is possible that the licorice caused the stomachache, the physician dismisses this as unlikely, since she already believes that the peanuts are causing the stomachache and this theory is sufficient to explain the new occurrence. As schematized in [Table 4.4](#), the physician’s recommendation, given the patient’s history of eating peanuts and getting stomachaches, is to avoid the peanuts but enjoy the licorice.



Dmitri Ma/Shutterstock.com

What questions should the doctor ask to determine if peanuts are the cause of his allergy?

In both of these examples, new information—or evidence—can be viewed in terms of its informational value—that is, how much new predictive value it gives us relative to what we already know or believe. For the stock investor, Herman offers no additional aid in predicting the market beyond what Doris already tells him. For the emergency room physician, the presence of licorice offers no new explanatory value in understanding the cause of the patient's stomachaches, given that the physician can already explain the problem as being caused by the peanuts. Research on learning has shown that other animals in addition to humans are similarly sensitive to the informational value of cues in determining which associations they do or do not learn.

Kamin's Blocking Effect

Compare the story of Doris and Herman to the earlier story of siblings Geeta and Sunil. In both cases, there was competition going on. Sunil beat out Geeta for their mother's attention because he shouted louder and was thus the more noticeable of the two children. In contrast, Doris beat out Herman for the stock-picking job not because she was louder, or even necessarily any better than Herman at the job, but because she got there sooner. From these stories, we see that there

are two ways to win a competition: by being more noticeable (i.e., louder) and by getting there sooner. These two situations mimic the two types of cue competition seen in conditioning paradigms. Competition based on salience is, as noted earlier, akin to what happens when overshadowing occurs in compound conditioning. But a form of overshadowing can also occur between two cues when the associative, or predictive, value of one of the cues is learned earlier than the other, as suggested by the story of Doris and Herman (and by the peanut and licorice example as well). Overshadowing due to temporal priority reflects a sensitivity to the informational value of a cue relative to another, co-occurring cue for making a prediction.

In the late 1960s, several studies of classical conditioning in animals pointed to the notion that for a potential CS to become associated with a US, the CS must provide valuable new information that helps an animal predict the future. Similarly, although a given cue may indeed predict a US, it may not become associated with that US if its usefulness has been preempted (blocked) by a co-occurring cue that has a longer history of predicting the US. Much as Doris's prior demonstration of her predictive value blocked the later hiring of Herman, and the patient's prior history of peanuts causing a stomachache blocked the physician from later believing that licorice was the cause, a prior-trained CS can block learning about another, redundant CS that is introduced later in training ([Kamin, 1969](#)).

In a classic study by Leon Kamin, rats were first trained that a light predicts a shock. Later on, they were trained that a compound stimulus of a light and tone also predicts the shock ([Kamin, 1969](#)). Kamin found that with this pattern of training, the rat will learn very little about the tone because the tone does not improve the rat's ability to predict the shock. The phenomenon is now formally known as **blocking**; it demonstrates that classical conditioning occurs only when a cue is both a useful and a nonredundant predictor of the future ([Table 4.5](#)).

TABLE 4.5 Kamin's blocking paradigm

Group	Phase 1	Phase 2	Phase 3 (test)
Control group	Rat sits in chamber; no training	Tone CS combined with light CS → shock US	Tone CS or light CS: medium CR
Experimental "pretrained" group	Light CS → shock US	Tone CS combined with light CS → shock US	Tone CS: little or no CR (learning is "blocked")

blocking

A two-phase training paradigm in which prior conditioning with one cue (CS1 → US) blocks later learning of a second cue when the two are paired together in the second phase of the training (CS1 + CS2 → US).

Kamin's original blocking study is worth describing in detail because of its influence on subsequent theories of learning. In this study, one group of rats (the control group) was trained with a compound cue consisting of a light and a tone; this cue was reliably followed by a shock (see [Table 4.5](#), control group, phase 2). The light and tone constituted a compound CS that the rats learned to associate with the shock US. Later, these rats would give a medium-strong CR to either the tone alone or the light alone, though not as strong a response as to both the light and tone together. This is an example of compound conditioning, as described earlier.

Consider, however, the behavior of Kamin's second group of rats, identified as the experimental, or pretrained, group in [Table 4.5](#). These rats first received pretraining in which the light by itself predicted a shock (phase 1). From this training, they learned to associate the light CS and the shock US. Next (phase 2), they were given training that paired the light-and-tone compound cue and the shock, just like the control group animals had received. However, unlike the control rats, rats in the pretrained group were already responding strongly to the light CS when they began the phase 2 compound training. For these rats, the additional presence of the tone provided no new information for predicting the US.

Phase 3 was a testing phase. When the pretrained rats were tested with the light alone, they continued to exhibit a strong CR to the light, much as they had at the end of phase 1. However, in phase 3, if they were tested with the tone alone, they would give almost no response at all. This suggests that they learned almost nothing about the relationship between the tone and the US, despite the compound training received in phase 2, in which the tone (combined with light) was repeatedly followed by the US ([Kamin, 1969](#)). In contrast, rats in the control group, which did not receive phase 1 pretraining, exhibited significant (albeit medium-strength) CRs in phase 3 to both the light by itself and the tone by itself. Thus, the blocking phenomenon, exhibited by the pretrained rats, can be summarized as follows: prior training of the light → shock association during phase 1 blocks learning of the tone → shock association during compound (light + tone) training in phase 2.

This is very similar to what happened with the emergency room physician. Having previously deduced that peanuts led to the earlier stomachache, the doctor failed to credit the licorice with any stomachache-inducing properties when the next tummy ache was preceded by both peanuts and licorice. For this physician, learning the peanut → stomachache association blocked learning

of any licorice → stomachache association (refer again to [Table 4.4](#)).

The Rescorla–Wagner Model of Conditioning

The blocking effect posed a challenge for simple theories of classical conditioning. It suggested that cues do not acquire strength solely on the basis of their individual relationships with the US; rather, *cues appear to compete with one another for associative strength*. Thus, in phase 2 of the blocking experiment in [Table 4.5](#), the tone competes with the light, and in the case of the pretrained group, the tone loses: since the light already accurately predicts the US, the tone provides no additional predictive information (just as Herman provides no additional value over Doris in predicting stocks and just as knowing that the patient ate licorice presents no additional value for predicting his stomachache if we already know that he ate peanuts; see [Table 4.4](#)).

The blocking paradigm demonstrated that contiguity of a cue and a US is not enough to elicit a CR, contrary to what Aristotle expected. For a stimulus to become associated with a US, it must impart reliable, useful, and nonredundant information ([Kamin, 1969](#); [Rescorla, 1968](#); [Wagner, 1969](#)). Apparently, “simple” Pavlovian conditioning is not as simple as psychologists once thought! In fact, rats (and other animals, including humans) appear to be very sophisticated statisticians. But how does one learn which are the most useful and informative cues to remember?

In the early 1970s, two Yale psychologists, Robert Rescorla and Allan Wagner, were independently trying to understand Kamin’s blocking effect and other related conditioning phenomena. Although the two researchers worked at the same university, they did not realize they were using the same approach to solve the same problem until they happened to be riding a train together to a conference and began chatting about their research. To their surprise, they discovered they had each come up with the same idea, and they decided to join forces ([Rescorla & Wagner, 1972](#)).

Seeking to understand how animals become aware of the informational value of stimuli, Rescorla and Wagner developed an elegantly simple way to formalize their approach. The key idea behind the Rescorla–Wagner model is that changes in CS–US associations on a trial are driven by the discrepancy (or error) between the animal’s expectation (or prediction) of the US and whether or not the US actually occurred. This error is sometimes referred to as the **[prediction error](#)**. How

one can learn by using prediction errors is described in the next section.

prediction error

The difference between what was predicted and what actually occurred.

Error-Correction Learning

“I have not failed,” said Thomas Edison, “I’ve just found 10,000 ways that won’t work.” Like Edison, the rest of us can, and do, learn from our failures. Consider Herman, who in our earlier example failed to be hired as a stock analyst. His real passion is tennis, and he hopes to have a future in that field. He practices hours each day, focusing especially on his serve. His goal is to put the ball as far back in the serving zone as possible but not so far that it goes past the line and is considered a fault.



Bill Losh/The Image Bank/Getty Images

Every time Herman serves too far outside the box, he learns to correct his swing so that next time he hits the ball not so far.
What kind of learning is Herman exhibiting?

On his first serve of the day, he puts the ball into the middle of the serving zone, where his opponent could easily return it. To make the next serve harder to return, he adjusts his stance a little, throws the ball higher, and swings harder. This time the ball lands closer to the end of the zone. On the third serve, he throws the ball a little higher still and hits it just a bit harder. This time, however, the ball goes too far; it is a fault. In response to this error, Herman again adjusts his serve. He throws the ball a little less high and hits it a little softer than the last time, achieving a perfect serve. After several tries, correcting his moves each time on the basis of how well he did previously, Herman has learned to hit a ball at just the right height and strength to make it land at the outer end of the service zone: not too close and not too far.

Herman has learned through a process of trial and error, using a method called **error-correction learning**, in which he uses the errors on each trial to make small changes in performance as he seeks to reduce the error on the next trial. Error-correction learning can be summarized by describing three situations that represent three types of outcomes Herman experienced: (1) when Herman hits the ball too close, he changes his next serve to hit the ball harder, so that it will land a little farther; (2) when Herman hits a perfect serve, he tries to do exactly the same thing the next time; and (3) when Herman hits the ball too far, he changes his next serve to hit the ball a little more softly, so it lands a little closer next time. The example of Herman perfecting his serve is an example of motor skill learning, which is discussed in more detail in [Chapter 8](#). But the same principle of error-correction learning applies more broadly to other types of learning, including classical conditioning.

error-correction learning

Learning through trial and error to reduce the discrepancy (error) between what is predicted and what actually occurs.

Rescorla and Wagner proposed that there are three key situations to consider when interpreting a prediction error, as summarized in [Table 4.6](#), and they are very similar to the three ways Herman learned from past outcomes to improve his tennis serve. One is a situation in which either no CS or a novel CS is presented, followed by a US, such that the US will be unexpected; this is considered a “positive” prediction error because there is more US than expected. The Rescorla–Wagner theory expects that the CS → US association should increase in proportion to the degree that the US is surprising; that is, the larger the error, the greater the learning. This makes sense because, if you failed to predict the US, you want to increase (move in a positive direction) your

likelihood of predicting it in the future (given the same CS). It is similar to what Herman does when his serve is too short and he positively increases the strength of his serve to send it farther next time.

TABLE 4.6 Error correction and response in the Rescorla–Wagner model and tennis

Conditioning error	R–W model response	Tennis error	Herman's response
Positive error: CS predicts nothing or too little, but US unexpectedly occurs or is unexpectedly strong	Increase association	Ball falls short	Increase strength of serve
No error: CS predicts US, and predicted US occurs	No new learning	Ball lands perfectly	Do same thing next time
Negative error: CS predicts US, but no US occurs	Decrease association	Ball goes too far	Decrease strength of serve

If, however, a well-learned CS is followed by the expected US, there is no error in prediction (the US was fully predicted by prior presentation of the CS), and thus no new learning is expected. This is similar to what happens when Herman makes a perfect serve: he doesn't want to change a thing from what he did last time. Finally, if the CS predicts a US, but the US does not occur, the prediction error is considered “negative,” and Rescorla and Wagner expect the trial to be followed by a decrease in the CS → US association. This is similar to what happens when Herman hits the ball too far and must reduce the strength of his serve next time.

Associative Weights and Compound Conditioning

The Rescorla–Wagner model assumes that each CS has an [associative weight](#), which is a value representing the strength of association between that cue and the US. In the blocking experiment described earlier and in [Table 4.5](#), there would be two associative weights: one for light and one for tone. Think of these weights as numbers that indicate how strongly the CS predicts the US. Before any training takes place, all associative weights are 0.0, meaning that when a potential CS first appears, there is no expectation that any US will follow. These associative weights change through learning as the animal discovers which stimuli predict the US and therefore which should have strong weights.

associative weight

In the Rescorla–Wagner model of conditioning, a value representing the strength of association between a conditioned stimulus (CS) and an unconditioned stimulus (US).

A critical property of the Rescorla–Wagner model is that the weights associated with one cue can indirectly influence the weights accruing to other, co-occurring cues. That is, if a tone and a light are both present on a trial, they will compete for associative strength (much as Doris and Herman competed for the stock analyst job). This competitive property of the Rescorla–Wagner model allows it to account for many important conditioning phenomena, especially those with complex stimuli involving multiple elements (such as tones and lights paired together). Most importantly, this cue-competition property of the Rescorla–Wagner model allows it to account for Kamin’s blocking effect, as described next.

Using the Rescorla–Wagner Model to Explain Blocking

Consider the simple network model of eyeblink conditioning diagrammed in [Figure 4.11](#). For every aspect of the real world that the model represents, the model contains a node—a small element that is activated, or “turned on,” when the network believes that a certain aspect of the world is or will be present. You can think of these nodes as being something like abstract neurons, or collections of neurons, which process information in a manner analogous to how activation flows through the brain. In the network model of [Figure 4.11](#), there is an input node for each CS (tone and light), an “actual output” node for the eyeblink CR, and a “teaching node” that shows whether or not the US (airpuff) actually occurred (indicating whether or not an eyeblink would have been appropriate). Thus, the teaching node shows the “desired output,” or what the CR should be. The three parts of [Figure 4.11](#) correspond respectively to the three phases of an experiment to demonstrate blocking.

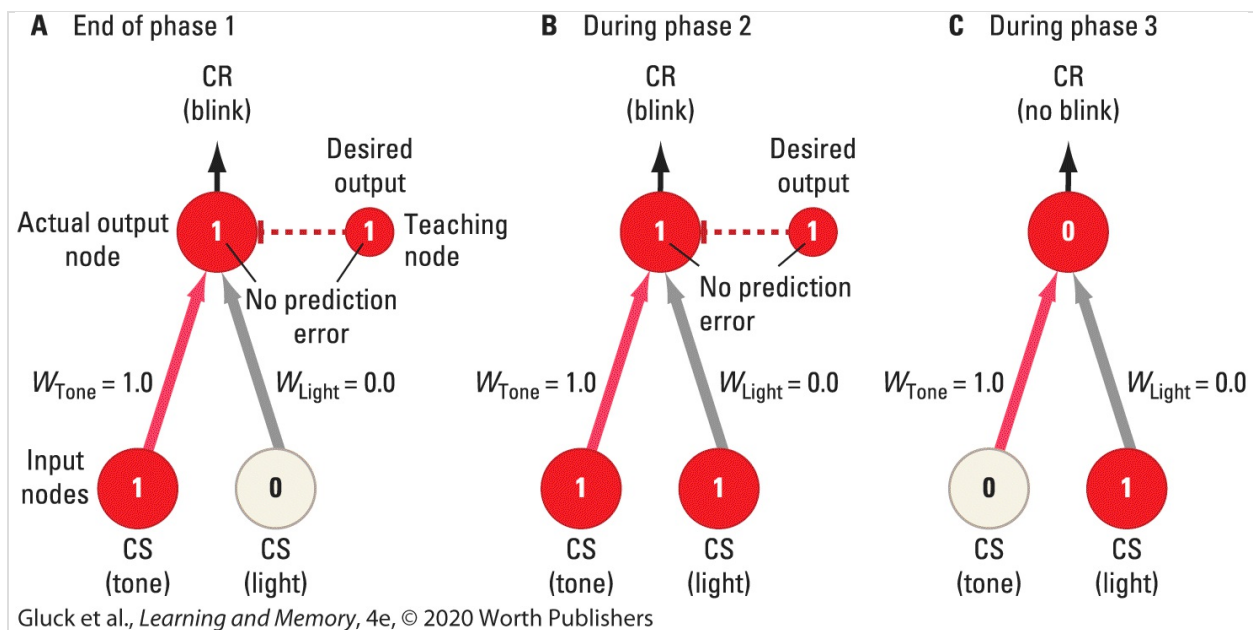


FIGURE 4.11 Blocking in the Rescorla–Wagner model (A) Phase 1 involves repeated pairing of the tone CS with an airpuff US. At the end of this phase, there is a strong associative weight for the tone input node, which in turn can activate the output node. (B) Phase 2 involves presentation of both tone and light CSs, paired with the US. Since the tone already perfectly predicts the US, causing the output node to be fully activated, there is no output prediction error, and no learning occurs. Hence, the weight from the light input node remains 0.0. (C) Finally, in phase 3, the network is tested with presentations of light alone. Since the associative weight of the light node was never changed from $W = 0.0$, there is no activation of the output node, and no behavioral response occurs. Hence, the Rescorla–Wagner model correctly shows blocking.

Description

The illustration labeled A, End of phase 1 shows repeated pairing of the tone C S with an airpuff U S. C S (Tone), weight 1.0, and C S (Light), weight 0.0 both have arrows pointing to C R (Blink), weight 1.0. Desired output 1.0 points to the C R.

The illustration labeled B, During phase 2 shows both tone and light C Ss, paired with the U S. C S (Tone), weight 1.0, and C S (Light), weight 1.0 both have arrows pointing to C R (Blink), weight 1.0. Desired output 1.0 points to the C R.

The illustration labeled C, During phase 3 shows the network is tested with presentations of light alone. C S (Tone), weight 0.0, and C S (Light), weight 1.0 both have arrows pointing to C R (Blink).

During phase 1, the network learns that a tone predicts an airpuff US. [Figure 4.11A](#) represents the state of affairs when a tone is presented at the end of phase 1 training. To show that the tone is presented, the tone input node is shaded red and given an activation value of 1. The other possible cue, a light, is off, so the light input node is not shaded at all and shows an activation value of 0. Thus, a solid fill color and a 1 in an input node means the CS is present; lack of color

and 0 in an input node means the CS is absent. In each trial in which the tone is paired with an airpuff, the teaching node is shaded red and given a value of 1, meaning the desired (or correct) response is occurrence of a blink.

Initially, at the start of phase 1 (not shown in [Figure 4.11](#)), there would have been a large prediction error on the first tone–airpuff trial because no learning would have yet taken place, and thus no blink would have occurred (the output would have been 0). However, after many pairings of the tone and airpuff, the network learned to give the correct actual output of a CR blink. This is why, in the diagram for the end of phase 1 ([Figure 4.11A](#)), the actual output node and the teaching node are both shown as fully active, with an activation value of 1 (and shaded dark red). This is a fully trained network that is responding consistently to the tone with an eyeblink CR. Because the response is correct, there is no prediction error: the actual output value of 1 equals the desired teaching output of 1. At this point (i.e., at the end of this tone–airpuff training), the tone has acquired an associative weight (W) of 1.0. In contrast, the light has an associative weight of 0.0 because the light was not presented during training.

Turn now to the start of phase 2 of our blocking experiment. Now the light and tone are presented together ([Figure 4.11B](#)), as shown by assigning both the tone and light input nodes in [Figure 4.11B](#) a value of 1. The actual output node receives activation equal to the sum of the associative weights accrued by all the active (present) cues, which in this case is $W = 1.0$ for the tone plus $W = 0.0$ for the light, for a total activation to the output node of 1.0 in [Figure 4.11B](#). This activation is sufficient to result in a blink. Since the compound cue in phase 2 will be followed by the airpuff US, the blink response is correct—and the prediction error is 0.0. Since weight change is determined by the prediction error and the prediction error is 0.0, there is no learning on this trial—or, indeed, on any other phase 2 trial. By the end of phase 2, the associative weights will still be the same as they were at the beginning of phase 2: the tone association is strongly weighted at $W = 1.0$, and the light has no associative weight ($W = 0.0$).

A subsequent test presentation of the light alone in phase 3 is shown in [Figure 4.11C](#): because there is no associative weight for the light input node, there is no activation of the output node—and therefore no response to the light alone. Thus, this simple graphical network model representation of the Rescorla–Wagner model shows Kamin’s blocking effect, just as was observed in the pretrained animals in [Table 4.5](#).

Compared to the experimental pretrained animals in Kamin's paradigm, the control rabbits ([Table 4.5](#)) get no training at all in phase 1, so the associative weights connecting potential cues to a potential US start at 0.0 and remain at 0.0 from start to end of phase 1. In phase 2, where the tone-and-light compound cue is paired with the US, there is a big prediction error (1.0) on the first trial when the US appears totally unexpectedly. Over the course of the training trials in phase 2, the associative weights for both the tone and light CSs will rise together until both are equally (but only mildly) associated with the US—say, to 0.5. But their sum will be enough together to predict the US, leading to an actual output value of 1.0 predicting the US by the end of phase 2 of the control-group training. In a subsequent testing phase (phase 3), members of the control group give a medium-strong response to either the tone or the light if either cue is presented individually because the associative weights for both tone alone and light alone are at 0.5, only half the strength required to predict the US (since they have always been presented together as a pair, with their associations combined and summed during phase 2).

As noted, Rescorla and Wagner's basic premise was that learning should occur in proportion to the degree to which the US is unexpected when it is experienced ([1972](#)). The Kamin blocking study is perhaps the clearest example of this influence of expectation (or prediction) on learning. A key assumption in the Rescorla–Wagner model is that when multiple CS cues are present, the expectation (or prediction) of the US is the sum of the associative weights of *all* of the cues present on that trial. Thus, Rescorla and Wagner argued, to understand the learning that takes place between a specific cue (CS) and an outcome of interest (the US), we must pay close attention to *all* the cues making up the context in which the training is conducted, especially the other cues that co-occur during learning.

Influence of the Rescorla–Wagner Model

More than four decades after its publication, the Rescorla–Wagner model is generally acknowledged as the most influential formal model of learning. Its broad acceptance is due to its elegant simplicity and to the fact that it explains a wide range of previously puzzling empirical results, such as the blocking experiments described previously. One hallmark of a successful model is that it reveals underlying connections between a series of observations that initially seemed unrelated or even contradictory.

The Rescorla–Wagner model also made surprising predictions about how animals would behave

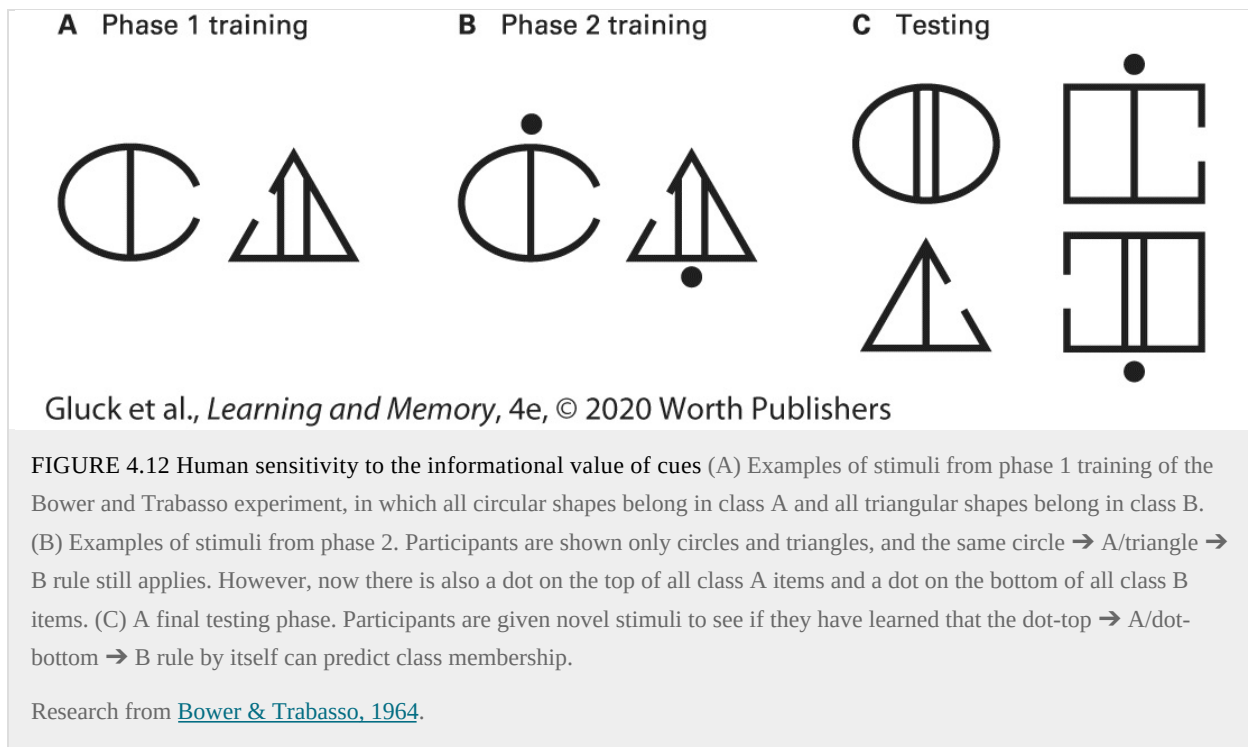
in *new* experimental procedures, and experimenters rushed to test those predictions. This is another feature of a successful model: it should allow scientists to make predictions that could not otherwise be foreseen without the model. Ideally, modeling and empirical work should constitute a cycle in which the model makes predictions that, when tested, provide new empirical data. If the data match the predictions, the model is supported. If not, the model must be revised. The revised model then generates new predictions, and the cycle continues.

Due to its simplicity, however, the Rescorla–Wagner model cannot account for every kind of learning—nor should it be expected to. Nevertheless, the Rescorla–Wagner model has been the starting point from which many other promising models have been built, including the models of human learning discussed next.

Error Correction in Human Category Learning

Do concepts such as blocking and models such as the Rescorla–Wagner model apply only to classical conditioning, or might they also provide insights into higher forms of human cognition and behavior, especially those that involve prediction or categorization?

Early research evidence for blocking-like effects in humans came from work by Gordon Bower and Tom Trabasso, who trained college students to categorize objects according to certain predefined rules ([Bower & Trabasso, 1964](#)). The students were presented with geometric figures varying in five dimensions: color, shape, number of internal lines, position of a dot, and position of a gap (noncolored examples are shown in [Figure 4.12](#)). Phase 1 of the experiment consisted of training the participants by asking them to guess whether each figure belonged to class A or class B; after each guess, they were told whether they had guessed correctly or not, which allowed them to perform error correction. For example, some participants were trained that all circular shapes belong in class A, while all triangular shapes belong in class B (no matter what other features the figures might have). (Two sample stimuli are shown in [Figure 4.12A](#), and the experiment is schematized in [Table 4.4](#), bottom row.) Given enough trials with different stimuli, participants would deduce the rule: circle → A/triangle → B.



Description

The illustration labeled A shows Phase 1 training. The shape on the left is a circle with a vertical bar and a piece missing from the perimeter on the right side. The shape on the right is a triangle with two vertical bars and a piece missing from the left side.

The illustration labeled B shows Phase 2 training. The shape on the left is like a circle with a vertical bar and a piece missing from the perimeter on the right side. It is topped by a solid dot. The shape on the right is a triangle with two vertical bars and a piece missing from the left side. A solid dot sits underneath it.

The illustration labeled C shows Testing. It is represented by four shapes as follows. The shape on the upper left shows a circle with two vertical bars. The shape on the lower left triangle has a vertical bar with a piece missing from its right side. The shape on the upper right shows a rectangle with one vertical bar and a piece missing from its right side. A solid dot is placed on top of the rectangle. The shape on the lower right shows a rectangle with two vertical bars and a piece missing from its left side. A solid dot is placed underneath the rectangle.

Once this lesson was mastered, the experimenter showed participants a slightly different set of figures: now all figures that were circular and thus belonged to class A had a dot on top, whereas all figures that were triangular and thus belonged to class B had a dot on the bottom ([Figure 4.12B](#)). This addition of a redundant cue (position of the dot) in phase 2 parallels the addition of the licorice stimulus in the second month of the physician trying to figure out what caused her patient's stomachache. Participants in the Bower and Trabasso study continued to perform well

by using their old rule of sorting on the basis of shape; the question was whether they would also learn that the dot position by itself predicted class membership.

To test this, the experimenters used new figures, shown in [Figure 4.12C](#). Given a figure with no dot, all participants continued to sort the circles into class A and the triangles into class B. However, when given a figure with a new shape (rectangle), none of the participants correctly sorted on the basis of dot position. Thus, these humans performed much like the physician (who dismissed the possibility that licorice could be causing her patient's discomfort), in that they displayed little or no response to the redundant cue added in phase 2. In effect, prior learning that the shape predicted class membership appears to have *blocked* subsequent learning that the dot position also predicted class membership. More recent studies have verified that this tendency to “tune out” information that is redundant with regard to previously learned cues is pervasive in many forms of human learning ([Kruschke, Kappenman, & Hetrick, 2005](#)).

Another example that links conditioning and cognition is a simple neural network model developed by Mark Gluck and Gordon Bower to model how people learn to form categories ([Gluck & Bower, 1988](#)). In this study, college students were asked to learn how to diagnose patients suffering from one of two nasty-sounding (but fictitious) diseases—midosis or burlosis. The students reviewed medical records of fictitious patients, who were each suffering from one or more of the following symptoms: bloody nose, stomach cramps, puffy eyes, discolored gums. During the study, each student reviewed several hundred medical charts, proposing a diagnosis for each patient as they examined the patient's chart and then being told the correct diagnosis ([Figure 4.13](#)). The students initially had to guess, but with practice, they were able to diagnose the fictitious patients quite accurately. The fact that the various symptoms were differentially diagnostic of the two diseases helped them improve. Bloody noses were very common in burlosis patients but rare in midosis, whereas discolored gums were common in midosis patients but rare in burlosis.

The patient is suffering from:

Bloody nose

Stomach cramp

What is your diagnosis?

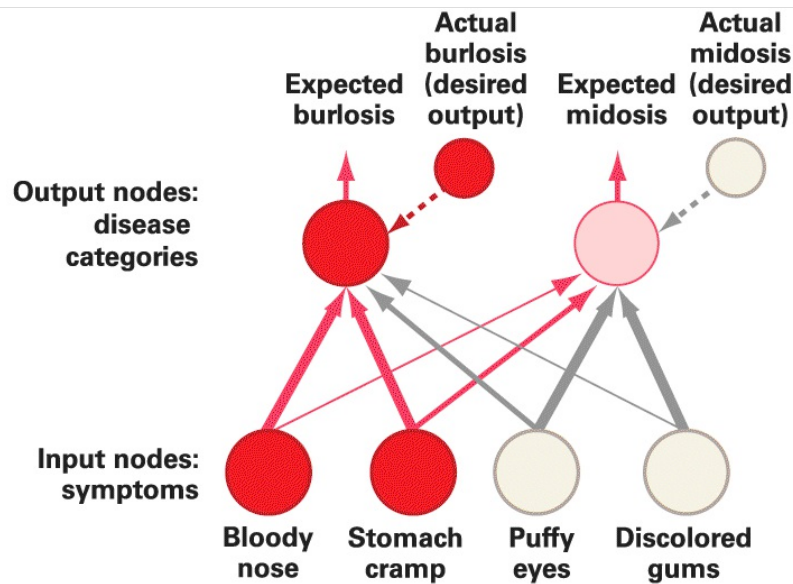
Burlosis

Midosis

Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

FIGURE 4.13 A sample training trial in Gluck and Bower's probabilistic categorization task. On a particular learning trial, a research participant would see a listing of symptoms (e.g., bloody nose and stomach cramp), make a diagnosis, and then be given feedback about whether or not the diagnosis was correct.

This kind of learning can be modeled using the network in [Figure 4.14](#). The four symptoms are represented by four input nodes at the bottom of the network, and the two disease categories correspond to the two output nodes at the top of the network. As learning progresses, the weights of the arrows leading from the symptoms to the diseases are updated according to the learning rule from the Rescorla–Wagner model, much as if the symptoms were CSs and the diseases were alternate USs. Thus, the Gluck and Bower disease-category learning model in [Figure 4.14](#) is, in principle, the same basic network model used by Rescorla and Wagner, shown in [Figure 4.11](#). The main difference is that the disease-category learning model has more possible outcome categories and more possible input cues.



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

FIGURE 4.14 Gluck and Bower's network model of category learning The arrows from bloody nose and stomach cramp to burlosis and from puffy eyes and discolored gums to midosis are thick, indicating highly diagnostic relationships (that is, heavily weighted cues). The other cues are of only moderate diagnosticity. This figure shows a trial in which a patient presents with two symptoms, bloody nose and stomach cramp; thus, these two input nodes are active (dark red). The other two input nodes represent symptoms that are not present (puffy eyes and discolored gums), and these nodes are inactive (gray). Relative activation levels (dark red and light red) of the two "expected" category nodes are based only on the weight of the input (the current associative weights of these cues) flowing up the arrows from the active and present symptoms (bloody nose and stomach cramp).

Information from [Gluck & Bower, 1988](#).

Description

The network model, represented using circles are divided into two sections- Output nodes- disease categories and Input nodes: symptoms. Thin and thick arrows depict the diagnostic relationships between the nodes.

The data given in the network model are as follows:

Output nodes: symptoms have two nodes- Expected burlosis and Expected midosis. Input nodes: symptoms has four nodes- Bloody nose, Stomach cramp, Puffy eyes, and Discolored gums.

Bloody nose and Stomach cramp have thick arrows, pointing to Expected burlosis. Puffy eyes and Discolored gums have thick arrows, pointing to Expected midosis. Bloody nose and Stomach cramp have thin arrows pointing to Expected midosis. Puffy eyes and Discolored gums have thin arrows pointing to Expected buriosis.

Learning and performance in the category learning model of [Figure 4.14](#) work as follows. As the subject learns the correct associations, the connections between nodes and disease categories

acquire weights that capture the true diagnosticity of each cue for each disease. For example, presentation to a research subject of the chart of a patient with the symptoms “bloody nose” and “stomach cramp” (as in the training trial illustrated in [Figure 4.13](#)) is modeled in [Figure 4.14](#) by turning “on” the corresponding input nodes, much as we turned on the tone node in [Figure 4.11](#). Activating these two input nodes causes simulated activity to travel up four weighted connections, shown as arrows: two to burlosis and two to midosis ([Figure 4.14](#)). On this trial, burlosis is the correct label, indicated by the very dark “actual burlosis” node, which is connected by a dashed line to the “expected burlosis” node (so the “actual burlosis” node represents the real truth about the patient, and the network is trained to predict, or generate, an expectation that matches that truth). The model is more likely to diagnose the patient as having the disease whose node receives the greater amount of activation—namely, burlosis—which in fact is the correct diagnosis.

By analogy with the Rescorla–Wagner model, these output-node activations are equivalent to the network’s *prediction* of one disease versus another. After a student guesses at a diagnosis and is told the correct answer, learning in that student is modeled by modification of the network’s weights so as to reduce future error, in accordance with the Rescorla–Wagner model’s error-correction learning rule. The network model shown in [Figure 4.14](#) incorporates nothing other than the learning principle of the Rescorla–Wagner conditioning model, yet this “animal conditioning” model of human cognition accounts for variations in how the participants classified different patient charts. The model correctly predicted the percentage of participants who would classify each of the 14 possible symptom charts as being midosis versus burlosis; it also predicted how well the participants were later able to make judgments about the probabilities of the two diseases when they knew only one of the symptoms.

Stimulus Attention and the Modulation of CS Processing

Despite the many successes of the Gluck and Bower model in predicting human cognitive data, several limitations of the model became evident in further studies of human category learning. In particular, as a model of category learning, it fails to account for people’s ability to actively focus their attention on one or another symptom (such as bloody nose *or* stomach cramp) or to shift or refocus this attention during learning. These limitations echo similar problems of the

Rescorla–Wagner model (on which the Gluck and Bower model is based)—specifically, the Rescorla–Wagner model’s inability to account for how attention to stimuli is modulated during learning, especially when animals or people are repeatedly exposed to stimuli that have no consequence.

To better understand where and how the Rescorla–Wagner model (and, by analogy, the Gluck and Bower model) falls short in accounting for attentional changes, let’s consider the example of Moira, the little girl at the beginning of the chapter who learned to run to the corner with money each time she heard the ice cream truck jingle in the distance. What do you think would have happened if, after Moira arrived in her new neighborhood, she continued to hear the jingle off in the distance each day, but the ice cream truck never appeared? If, weeks later, the truck did begin to appear after the jingle was heard, do you think Moira would quickly learn to again associate the jingle in the distance with the forthcoming arrival of the ice cream truck? Prior learning that a cue is irrelevant (like the jingle without the ice cream truck appearing) retards our ability to learn later that the cue has acquired some new predictive or associative relevance.

This learning about a cue’s irrelevance through exposure to the cue alone (with no associated significant event) is known as **latent inhibition**, a reduction in learning about a stimulus (CS) to which there has been prior exposure without any consequence (that is, no US). The name refers to the fact that the exposure latently (that is, implicitly) appears to inhibit later learning about the cue. This phenomenon was first described in the animal conditioning literature by Robert Lubow and Ulrich Moore ([Lubow & Moore, 1959](#)). Lubow and Moore’s study was conducted using sheep and goats; however, for consistency with the rest of this chapter (and to facilitate comparison with other studies previously discussed), we will describe their latent inhibition paradigm using rabbit eyeblink conditioning, which has reliably produced the same results.

latent inhibition

A conditioning paradigm in which prior exposure to a CS retards later learning of the CS–US association during acquisition training.

Latent inhibition studies use two groups of subjects: the first group (the control group) receives no pretraining, whereas the second group does receive pre-exposure training, as summarized in [Table 4.7](#). Control animals simply sit in their chambers until they are ready for the critical phase 2, in which they are trained to associate a tone CS with an airpuff-in-the-eye US. In contrast, animals in the pre-exposed group are repeatedly exposed to a tone with no US in phase 1 before

they undergo the same tone training in phase 2 as the control animals do. Thus, the only difference between the two groups is that one group is pre-exposed to the tone in phase 1.

TABLE 4.7 The latent inhibition paradigm

Group	Phase 1	Phase 2
Control group	Animal sits in chamber	Tone CS → airpuff US
Experimental “pretrained” group	Tone CS presented (but not US)	Tone CS → airpuff US

As illustrated in [Figure 4.15](#), rabbits in the pre-exposed group learn to associate the tone with a puff of air much more slowly in phase 2 than do rabbits in the control group ([Shohamy, Allen, & Gluck, 2000](#)). The same kind of slow learning following CS pre-exposure is seen in a variety of species and is also seen in human eyeblink conditioning ([Lubow, 1973](#)).

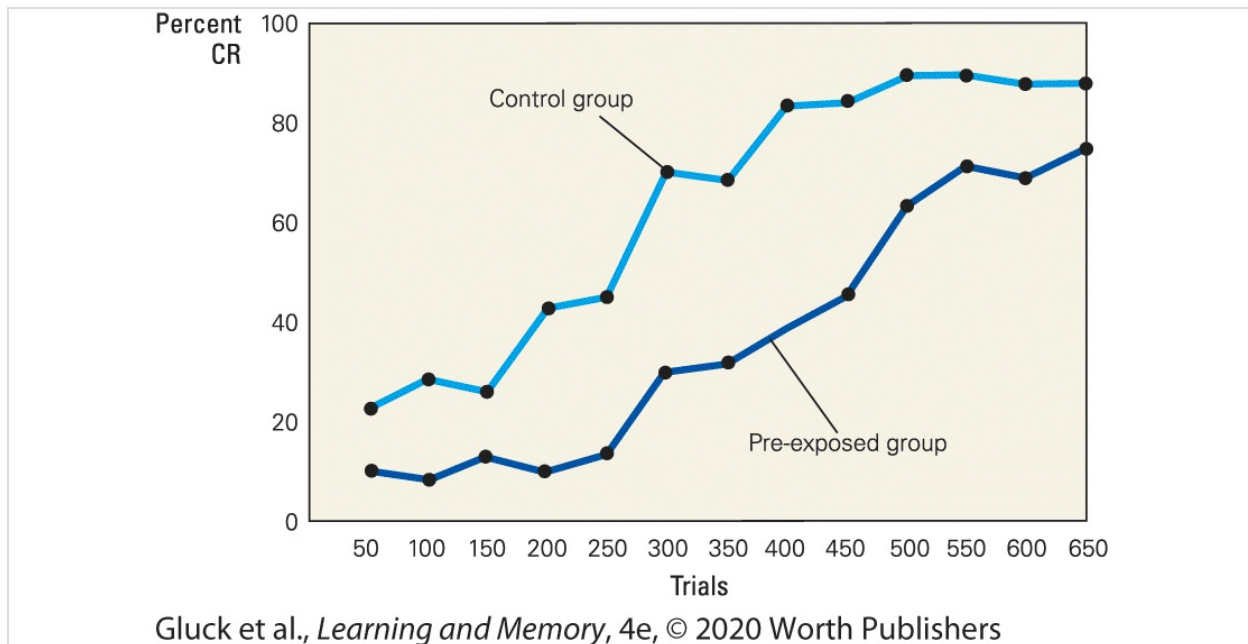


FIGURE 4.15 Latent inhibition in rabbit eyeblink conditioning This graph shows the percentage of trials that produced CRs in each block of 50 trials during the common phase 2 tone–airpuff training of the rabbits in the study. The rabbits in the control group learned rapidly. In contrast, the rabbits in the pre-exposed group, which had previously experienced 850 trials of tone-alone presentations in phase 1, learned much more slowly.

Data from [Shohamy et al., 2000](#).

Description

The X-axis plots Trials, with values ranging from 50 to 650 at an increment of 50 units. The Y-axis plots Percent C R, with values ranging from 0 to 100, at an increment of 20 units. The data provided in the graph are as follows:

The Pre-exposed group: (50,10), (100, 9), (150, 11), (200, 10), (250, 13), (300,22) (350, 25), (450,35), (500, 60), (550, 65), (600,60) (650, 65).

The Control group: (50, 20), (100, 25), (150, 21), (200, 40), (250, 42), (300, 65), (350, 63), (400, 80), (450, 81), (500, 82), (550, 84), (600, 83), (650 85).

Latent inhibition—that is, impaired learning following cue pre-exposure—is problematic for the Rescorla–Wagner model: there is no surprise during the first phase of tone-alone exposure and thus no prediction error. Therefore, the Rescorla–Wagner model expects no learning to occur in phase 1. As a consequence, the Rescorla–Wagner model makes the incorrect prediction that the pre-exposed group should be no different from the control group at the start of phase 2, a prediction clearly disconfirmed by Lubow’s studies, as well as by the data in [Figure 4.15](#).

Latent inhibition and similar paradigms that involve learning during mere exposure to apparently neutral cues suggest that there is more going on during conditioning than the error-driven learning characterized by the Rescorla–Wagner model. [Chapter 3](#) introduces some of these non-associative mechanisms, such as habituation and sensitization. To account for latent inhibition and other phenomena beyond the scope of the Rescorla–Wagner model, several alternative theories of conditioning have been proposed, and these are described next.

An Attentional Approach to Stimulus Selection

The Rescorla–Wagner model is often called a [US modulation theory](#) of learning because it proposes that the manner in which the US is processed determines what stimuli become associated with that US. Thus, in the Rescorla–Wagner model, the ability of the US to promote learning is modulated by how unexpected the US is, given the potential CS that precedes it. The error-correction principle of learning described earlier—the core idea behind the Rescorla–Wagner model—is that error modulates the ability of a US to promote learning about the CSs that preceded it.

US modulation theory

Any of the theories of conditioning that say the stimulus that enters into an association is determined by a change in how the US is processed.

An alternative class of learning theories focuses instead on the CSs, suggesting various mechanisms that modulate (either increase or reduce) the ease with which potential CSs can be

incorporated into an association. For this reason, they are referred to as **CS modulation theories**: they propose that the way attention to different CSs is modulated determines which CSs become associated with the US. One such theory, presented by Nicholas Mackintosh in the early 1970s, is based on the observation that people and animals have a limited capacity for processing incoming information ([Mackintosh, 1975](#)). This limited capacity means that paying attention to one stimulus diminishes (and hence modulates) our ability to attend to other stimuli.

CS modulation theory

Any of the theories of conditioning that say the stimulus that enters into an association is determined by a change in how the CS is processed.

Remember the blocking analogy in which Doris was the first to establish herself as a reliable predictor of the stock market, such that later, when Herman showed up, you gave him little credit for making equally successful predictions? (See also [Table 4.4](#).) The Rescorla–Wagner model argues that this outcome is due to the stock market (the US) already being well predicted by Doris (the first CS), so that no additional value (no learning) is attached to Herman (a potential second CS). However, Mackintosh’s view of blocking is quite different. He argues that you come to devote all of your attention to Doris because she has a long history of predicting the stock market, and thus you have no attention left to pay to Herman. The core idea of the Mackintosh theory is that a previously conditioned stimulus derives its salience from its past success as a predictor of important events ([Mackintosh, 1975](#)), and this happens at the expense of other, co-occurring cues that do not get access to your limited pool of attention. In essence, Rescorla and Wagner’s model lets Herman come in for an interview but doesn’t consider him valuable for predicting the market, while Mackintosh’s model never lets Herman in the door.

We turn next to review one of the situations in which Mackintosh’s CS modulation theory does a better job of explaining some aspects of learning phenomena than does Rescorla and Wagner’s US modulation theory.

An Attentional Explanation of Latent Inhibition

Recall that the Rescorla–Wagner model cannot explain cue–pre-exposure phenomena such as latent inhibition because, as a US modulation theory of learning, it only explains learning that takes place when a US is present or when previously trained cues predict the US. Thus, the Rescorla–Wagner model suggests incorrectly that no learning takes place when a neutral

(previously untrained) cue is presented. In contrast, Mackintosh's model predicts that the salience of a tone as a potential CS will decrease when the tone is presented without any US because the tone develops a history of predicting nothing. According to Mackintosh, the animal treats these tone-alone trials as if they were the little boy who cried wolf. Eventually the tones (like the boy) are ignored because they do not reliably predict that anything bad or good is about to happen.

Although these CS modulation models have had many successes—especially in clarifying behavioral phenomena that are not explained by the Rescorla–Wagner model—they have had less impact on the field of learning and memory, in part because they are more complex than the Rescorla–Wagner model and because they do not explain as broad a range of behaviors. Moreover, as discussed earlier in this chapter, the Rescorla–Wagner model has been especially influential because it works on the same fundamental principle as the learning algorithms employed in the connectionist network models of human memory used by cognitive psychologists, including the models of David Rumelhart and colleagues described in [Chapter 1 \(Rumelhart & McClelland, 1986\)](#) and the category learning model of [Gluck and Bower \(1988\)](#) discussed previously.

Which view is correct: the CS modulation or the US modulation approach to conditioning? For many years, the two camps were viewed as being in direct conflict, with each entrenched on a different side of the Atlantic Ocean: the US modulation view predominated in the United States (where Rescorla and Wagner worked), while the CS modulation view predominated in the United Kingdom (where Mackintosh and colleagues worked). However, behavioral and biological studies of conditioning now suggest that *both* views are probably correct; that is, there are likely to be both CS modulation and US modulation mechanisms engaged in learning. As you will see in [Section 4.2](#), part of what has helped resolve this debate is new data from neuroscience that have identified differential neural substrates for these two types of learning processes. This is one more example of the many areas where new forms of data from neuroscience have informed and helped resolve long-standing questions in psychology.

TEST YOUR KNOWLEDGE

Contrasting the Rescorla–Wagner and Mackintosh Models

1. Fill in the blanks: The Rescorla–Wagner model explains conditioning as modulation of the effectiveness of the _____ for learning, while the Mackintosh model explains conditioning as occurring through modulation of

attention to the _____.

2. From the following examples, which explanation of Connie's behavior would be best explained by the Rescorla–Wagner model? Which would be better explained by the Mackintosh model?
 - a. Connie loved the oatmeal raisin cookies so much that she devoted all of her attention to them. She didn't even bother tasting the chocolate chip cookies.
 - b. Connie was happy eating only the oatmeal raisin cookies, and she didn't feel any need to begin eating a new type of cookie.

(Answers appear at the end of the chapter.)

Other Determinants of Conditioning

Both the US modulation model of Rescorla and Wagner and the CS modulation model of Mackintosh have been influential in enhancing our understanding of associative learning ([Mackintosh, 1975](#); [Rescorla & Wagner, 1972](#)). They are powerful models precisely because they boil down the behavioral process of learning to its essential elements so that we can see the underlying, fundamental principles at work. However, as a result of such simplification, these models necessarily ignore many of the more subtle facets of conditioning, such as the role of timing and the importance of innate biases for associating different stimulus cues.

Timing

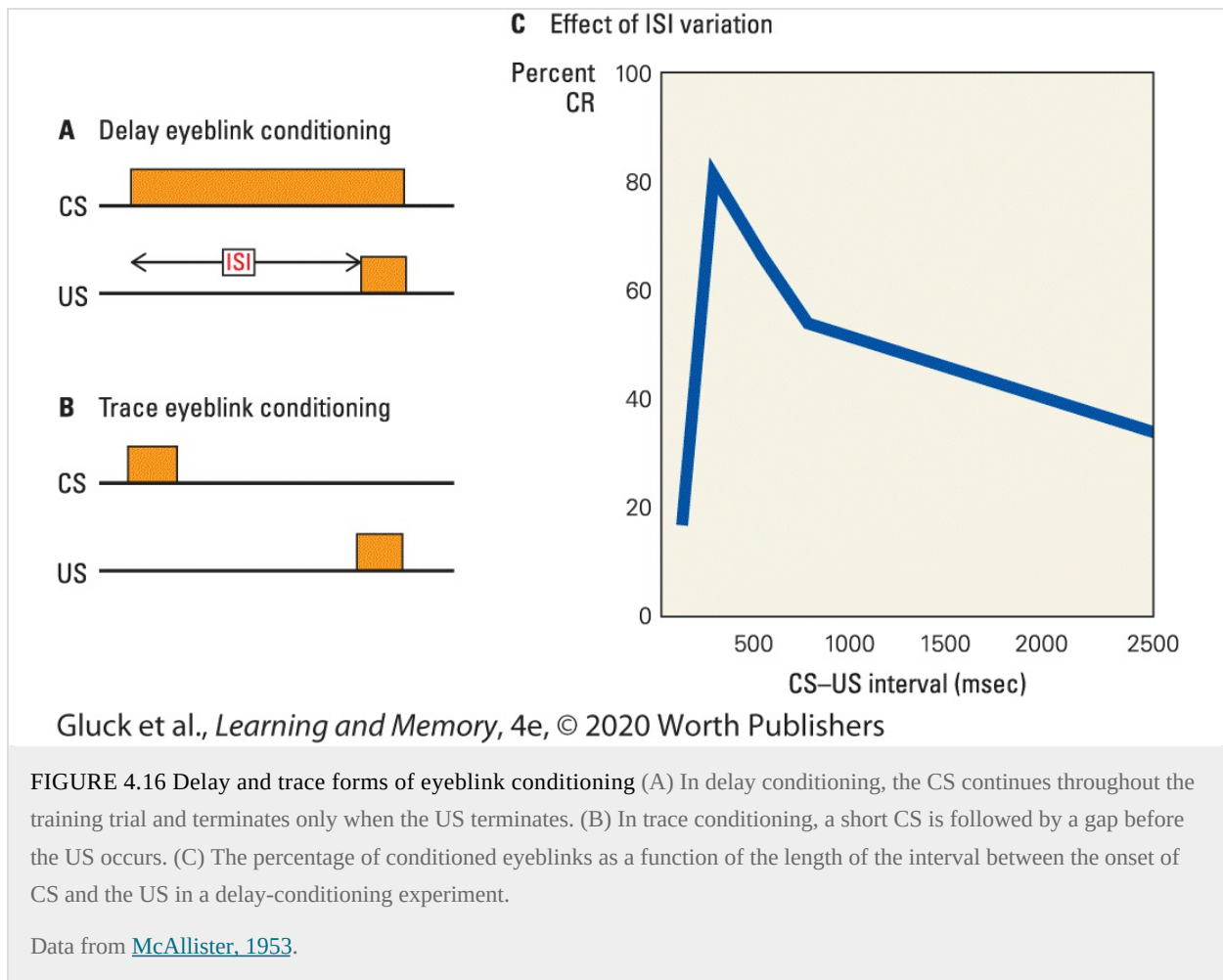
The Rescorla–Wagner model and the Mackintosh model both treat classical conditioning as if it were always composed of a series of discrete trials that occur one after the other. Moreover, these **trial-level models** treat each trial as a single event that results in a single change in learning. In reality, conditioning is more complex, and a trial consists of many events that can vary in different ways from trial to trial. For example, these models do not describe the timing of the animal's response within a given trial: does the CR occur right after the CS begins, or is it delayed until just before the US occurs? This information is lost in a trial-level model that only describes the aggregate effect of a training trial in terms of an overall association strength. Thus, one cost of having a simple and powerful model is that it cannot account for every detail of the animal's behavior.

trial-level model

A theory of learning in which all the cues that occur during a trial and all the changes that result are considered a single event.

One important aspect of many conditioning studies is the temporal relationship between the CS

and the US. **Figure 4.16A** illustrates eyeblink conditioning that is conducted using an approach known as **delay conditioning**, in which the tone CS continues throughout the trial and ends only once the US has occurred. (This is, in fact, how all of the animals were trained in the rabbit eyeblink-conditioning studies reported so far in this chapter.) The term *delay* refers to the delay from time of onset of the CS to onset of the US (as opposed to their occurring simultaneously).



Description

The illustration labeled A shows Delay eyeblink conditioning. In the two stimulus applications, the U S occurs at the end of an extended period of C S.

The illustration labeled B shows trace eyeblink conditioning. In the two stimulus applications, the C S occurs briefly, then there is a delay, then the U S occurs briefly.

The illustration labeled C a line graph that plots Effect of ISI variation. The X-axis shows Percent C S and Y-axis shows C S_ U S interval. The data in the graph shows that the percent of conditioned responses declines with an increased delay. It peaks with a delay below 500 milliseconds but quickly drops off as the delay lengthens.

delay conditioning

A conditioning procedure in which there is no temporal gap between the end of the CS and the beginning of the US and in which the CS co-terminates with the US.

Another form of conditioning that also includes an interval between CS and US, called **trace conditioning**, is represented in [Figure 4.16B](#); it uses a shorter CS that terminates some time before the onset of the US, requiring the animal to maintain a memory “trace” of the CS to associate with the subsequently arriving US. Although many trial-level learning models treat these types of conditioning as if they were equivalent, many studies have shown that learning behaviors, and the neural substrates associated with them, can be quite different for trace and delay-training procedures.

trace conditioning

A conditioning procedure in which there is a temporal gap between the end of the CS and the beginning of the US.

Even within a simple delay-training procedure such as that shown in [Figure 4.16A](#), variations in the **interstimulus interval (ISI)**, the temporal gap between the onset of the CS and the onset of the US (indicated in [Figure 4.16A](#)), can have significant effects. For eyeblink conditioning in the rabbit, the optimal ISI for fastest learning is about one-quarter of a second (250 msec), as shown in [Figure 4.16C](#). Shorter or longer intervals make learning more difficult for the animal and necessitate additional training trials. One of the remarkable aspects of rabbit eyeblink conditioning is that the timing of the CR corresponds exactly to the ISI (see [Figure 4.7](#), day 5), such that the eyelid is closed at precisely the moment the onset of the US is expected. The need to better understand the role of timing in learning is one of the challenges at the forefront of current learning research.

interstimulus interval (ISI)

The temporal gap between the onset of the CS and the onset of the US.

Associative Bias

The formal learning models described thus far imply that any arbitrary cue (such as a tone or a light) can be associated with any outcome, be it a shock or food. But is that really true? Consider Mandy, who came down with a strange illness soon after eating escargot (snails) at dinner. The same evening that she ate the snails, she also went to the movies with her date and saw a romantic comedy. Later that night, she woke up with a fever and a terrible case of hives. Both

eating the escargot and watching the romantic comedy were events that preceded the illness. But whereas Mandy has had an aversion to eating escargot since that evening, she has not stopped going to see romantic comedies. What this suggests is that not all cues are equally likely to be associated with every outcome. Rather, it appears that there is an *associative bias* whereby some cues (such as food) are more likely to be associated with some outcomes (such as illness).



Mark Gluck

If Mandy gets sick the next morning, will she blame the escargot or the movie she saw? What does that illustrate?

This bias was strikingly demonstrated in a study of [conditioned taste aversion](#), in which subjects learn to avoid specific tastes ([Garcia & Koelling, 1966](#)). John Garcia and R. A. Koelling trained rats with compound stimuli consisting of an unfamiliar taste and an unfamiliar tone (a rat’s version of watching a romantic comedy while eating snails). One group of rats was then injected with a poison that made them ill; a second group of rats was given an electric shock instead ([Table 4.8](#)). Which cue would the rats in each group “blame” for their illness or shock: the taste or the tone stimulus? To see which cues were most readily associated with which outcomes, the experimenters subsequently tested the rats with each of the cues independently: on some test trials, the rats were given food with the same novel taste but no tone, whereas on other test trials, the rats were presented with the tone but no food.

conditioned taste aversion

A conditioning preparation in which a subject learns to avoid a taste that has been paired with an aversive outcome, usually nausea.

TABLE 4.8 The Garcia–Koelling taste-aversion study

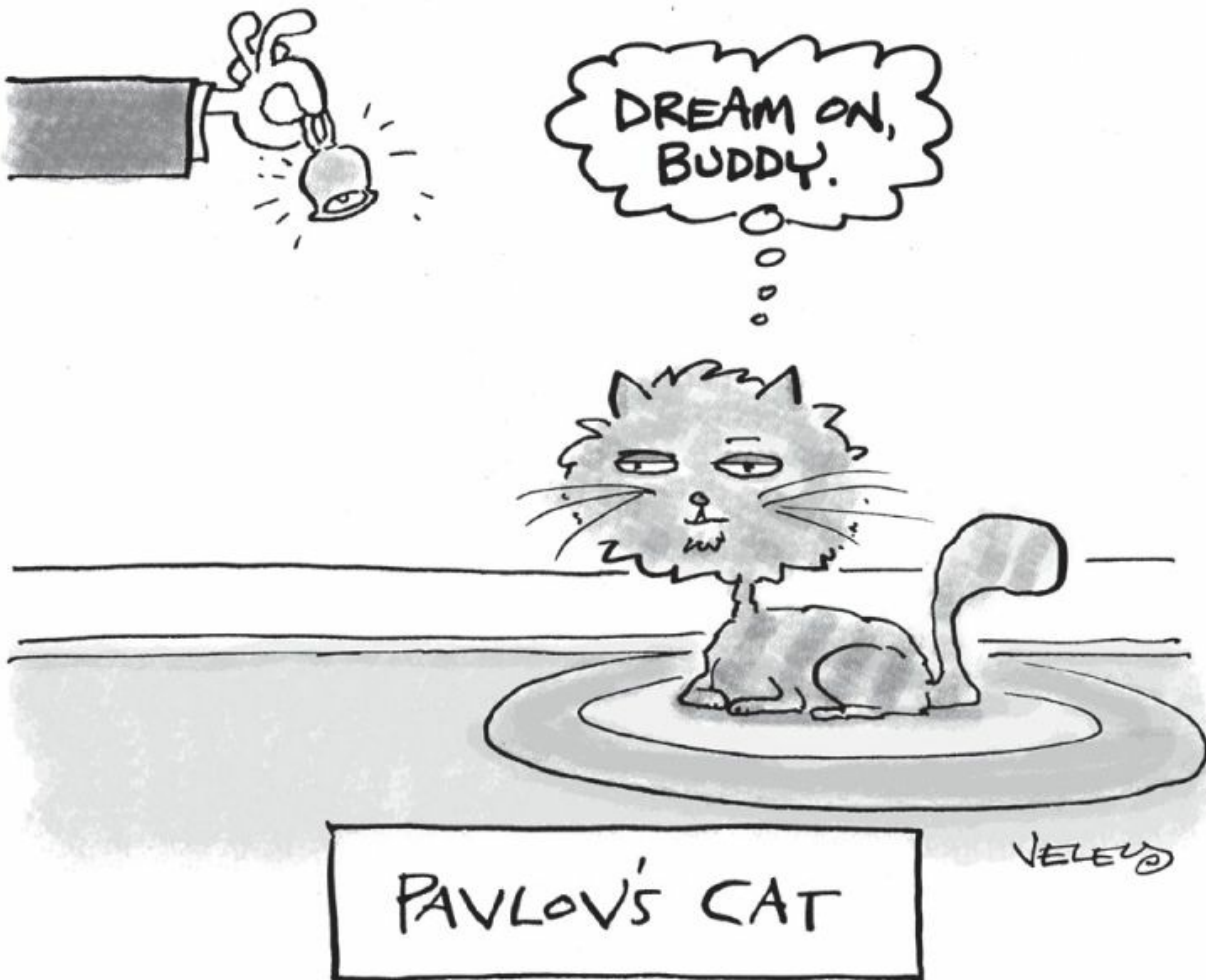
Group	Phase 1	Phase 2
Poison group	Tone + taste → poisoning	Tone → weak CR
		Taste → strong CR
Shock group	Tone + taste → shock	Tone → strong CR
		Taste → weak CR

The researchers found that the rats in the poison group were far more likely to associate the taste stimulus with their illness than to associate the tone with their illness (much as Mandy would be more likely to blame snails rather than romantic comedies for her illness). In contrast, the rats in the shock group were more fearful in the presence of the tone stimulus than when they encountered the taste stimulus. Garcia and his colleagues concluded that taste is a more effective stimulus for learning to predict illness but that an audio cue is more effective for learning to predict a shock. Clearly, rats, like people, have prior biases about what should predict what. This is not to say that you couldn’t be trained to throw up at romantic comedy movies, but it would be much harder (and require more training) than Mandy learning to avoid the taste of escargot.

Remember the quail that were trained to associate a light with sex? Although the quail were able to learn this association following many trials of training, Domjan and colleagues found that

quail could be conditioned much faster and more robustly if the CS, rather than being an arbitrary cue like a light, were something naturally associated in the wild with available females. These cues included the sight of a female at a distance or the sight of a female's head when the rest of her body is hidden in the underbrush ([Cusato & Domjan, 1998](#)).

Why are both Mandy and Garcia's rats more likely to associate food, rather than other cues, with getting sick? The answer may have to do with the potential *causal* relationship between eating food and getting sick that is a very real part of a person's or another animal's natural environment. In contrast, there is unlikely to be a natural causal relationship between watching a movie (good or bad) or hearing a tone and getting sick. Perhaps a sensitivity to the likely causal relationships is what guides and biases associative learning in animals. The best predictors of future events are the causes of those events, or at least their detectable indicators ([Dickinson, 1980](#)). Thus, it would make evolutionary sense for humans and other animals to be biased toward learning associations that correspond to causal relationships in the ecological niches in which the animals live and evolve.



Bradford Veley/www.CartoonStock.com

Suggest a CS and US Pavlov could have used instead of bells and meat if he had chosen cats rather than dogs for his experiments.

Interim Summary

- Classical conditioning involves learning about the predictive nature of stimuli in the environment—that is, what cues predict desirable or undesirable events. If an unconditioned stimulus (US) is repeatedly and reliably preceded by a neutral stimulus, such as a bell, that neutral stimulus can become a conditioned stimulus (CS) that evokes an anticipatory response, called the conditioned response (CR). CRs are anticipatory responses that prepare an animal for the expected US.
- The pairing of a potential CS with a US is not sufficient for conditioning to occur. Rather, for a CS to become associated with a US, it must provide valuable new information that

helps an animal predict the future. Even if a given cue is predictive of a US, it may not become associated with that US if its usefulness has been preempted (“blocked”) by a co-occurring cue that has a longer history of predicting the US.

- [Rescorla and Wagner \(1972\)](#) argue that learning should occur in proportion to the degree to which the US is unexpected when it is experienced. A key assumption in the Rescorla–Wagner model is that when multiple CS cues are present, the expectation (or prediction) of the US is the sum of the associative weights of all the cues that are present on that trial.
- CS modulation theories of learning (like Mackintosh’s model) presume that limits in attentional capacity cause our attention to one stimulus to decrease our ability to attend to other stimuli. In contrast, the Rescorla–Wagner model is a US modulation theory of learning because it sees the learning of associations as depending on how accurately the US is predicted based on all available information. Current behavioral and biological studies of conditioning now suggest that both CS modulation and US modulation mechanisms are likely to be involved in learning.
- Taste is more effective than an audiovisual stimulus for learning to predict illness, whereas an audiovisual cue is more effective for learning to predict a shock. One interpretation of this difference is the presence of an associative bias that favors potential causal relationships found in the animal’s natural environment, such as the potential relationship between eating food and getting sick.

4.2 Brain Substrates

Pavlov was a physiologist. When he discovered associative learning in his dogs in the early 1900s, he was naturally interested in understanding the brain mechanisms responsible for it. He even conducted a few experiments to examine how cortical lesions affect conditioning. However, in Pavlov's time, the technology for observing the brain's inner workings was not highly developed. Only in recent years have scientists gained knowledge and techniques that allow detailed study of the neural circuits for conditioning. We review here two neural systems—one in mammals and the other in invertebrates—that illustrate how studies of the neural bases of conditioning have yielded insights into the circuits, cells, molecules, and genes controlling the formation of new memories.

Conditioning of Motor Reflexes in the Mammalian Brain

As you can see in [Figure 2.2](#), the [cerebellum](#) sits just behind and slightly below the rest of the brain and looks like a miniature brain itself. In fact, the name *cerebellum* is Latin for “little brain.”

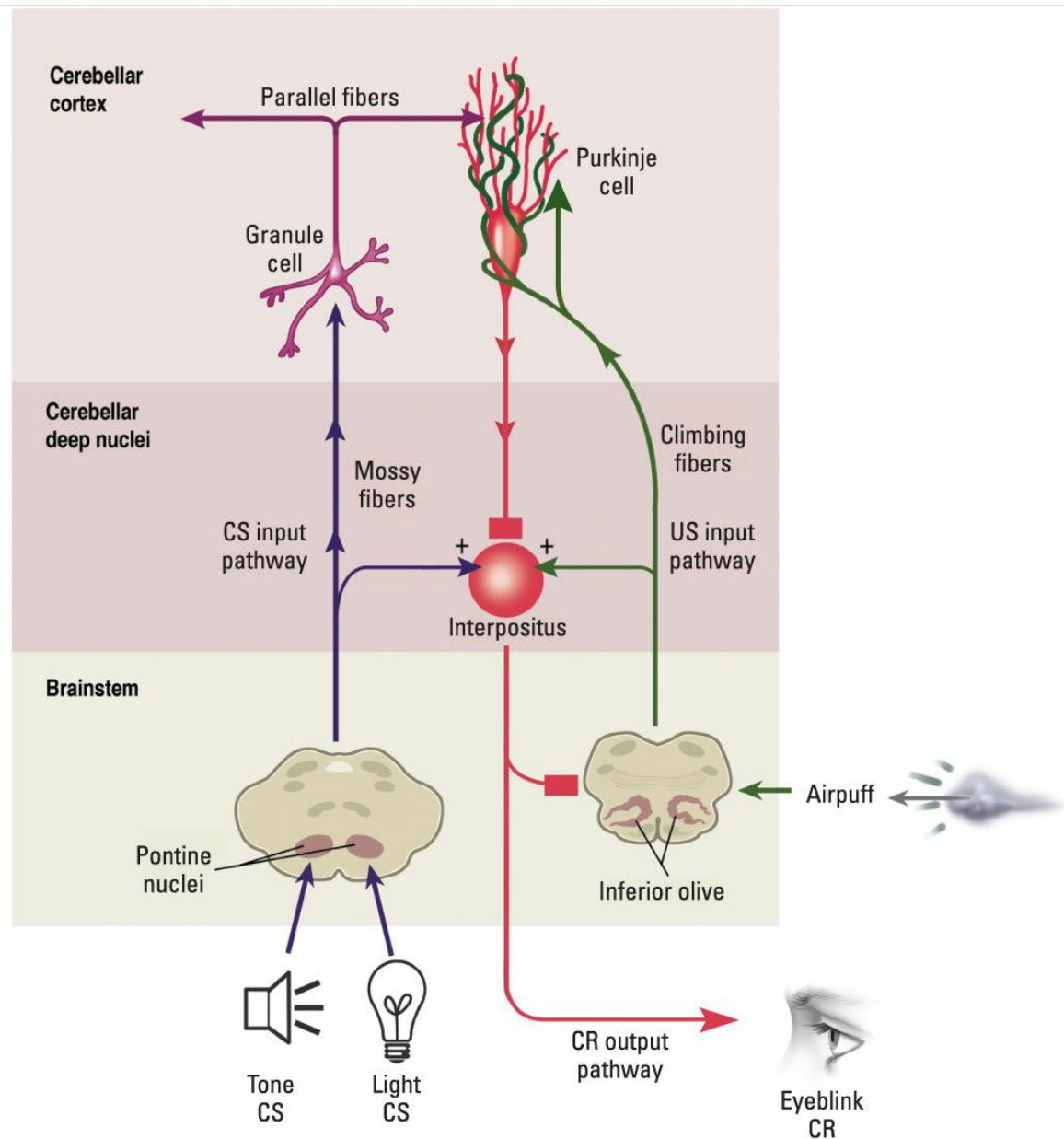
cerebellum

A brain region that lies below the cerebral cortex in the back of the head. It is responsible for the regulation and coordination of complex voluntary muscular movement, including classical conditioning of motor-reflex responses.

In the early 1980s, Richard Thompson and his coworkers made a startling discovery: small lesions in the cerebella of rabbits permanently prevented the acquisition of new classically conditioned eyeblink responses and abolished retention of previously learned responses ([Thompson, 1986](#)). Subsequently, Thompson and his colleagues studied the cerebellum and its role in motor-reflex conditioning for more than 25 years. Their work provides an instructive example of how support for a theory can be strengthened by converging evidence from a variety of scientific methods, such as electrophysiological recordings, brain stimulation, experimental lesions, temporary inactivation of brain structures, and genetically engineered animals ([Thompson & Steinmetz, 2009](#)).

The cerebellum has two main regions, as diagrammed in [Figure 4.17](#). Lying along its top surface is the *cerebellar cortex*, which contains certain large, drop-shaped, densely branching neurons

called **Purkinje cells**. Beneath the cerebellar cortex lies a collection of cells called the *cerebellar deep nuclei*, one of which is the **interpositus nucleus**. There are two major sensory-input pathways to the cerebellum: the CS input pathway and the US input pathway. The CS input pathway is shown in purple in [Figure 4.17](#). (Not all types of cells in the cerebellum are shown here; the figure shows only the cells and pathways that are critical for understanding the cerebellar circuits for motor-reflex conditioning.) CS pathways from elsewhere in the brain project (that is, send axonal projections) first to an area in the brainstem called the *pontine nuclei*. The pontine nuclei have different subregions for each kind of sensory stimulation. Thus, a tone CS would travel to one area of the pontine nuclei and a light CS to another. This CS information then travels up to the deep nuclei of the cerebellum along axon tracts called the *mossy fibers*, which branch in two directions. One branch makes contact with the interpositus nucleus. The other branch projects up toward the cerebellar cortex (by way of the granule cells and other cells not shown) and across the parallel fibers, and it connects to the dendrites of the Purkinje cells.



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FIGURE 4.17 Cerebellar circuits for motor-reflex conditioning in mammals A schematic diagram of the cerebellar circuits for conditioning. The CS input pathway is purple, the CR output pathway is red, and the US input pathway is green. Excitatory synapses are shown as arrows, and each inhibitory synapse terminates with a rectangle.

Description

The pathway begins with Tone C S and Light C S. It points to the oval-shaped Pontine nuclei, in the brainstem region. Further, the C S input pathway enters the cerebellar deep nuclei region, with Mossy fibers. Gradually, passes the granule cell in the Cerebellar cortex region and meets the parallel fibers. The parallel fibers branch bidirectional to the cerebellar cortex region and purkinje cells. The C R output pathway begins at the Purkinje cell. It passes to meet the

Interpositus region and results in Eyeblink C R.

When Airpuff is introduced, it passes through the Inferior olive region in the brainstem and the climbing fibers steer upward the U S input pathway and meet the Purkinje cells, which further moves downward the C R output pathway and results in eyeblink C R.

Purkinje cell

A type of large, drop-shaped, and densely branching neuron in the cerebellar cortex.

interpositus nucleus

One of the cerebellar deep nuclei from which the conditioned response output is generated in classically conditioned motor responses.

The second sensory-input pathway, shown in green, is the US pathway. An airpuff US to the eye activates neurons in the [inferior olive](#)—a structure in the lower part of the brainstem—which in turn activates the interpositus nucleus. In addition, a second branch of the pathway from the inferior olive projects up to the cerebellar cortex by means of the climbing fibers ([Figure 4.17](#)). Each climbing fiber extends to and wraps around a Purkinje cell. The climbing fibers have a very strong excitatory effect on the Purkinje cells, indicated in [Figure 4.17](#) by the large arrowhead at this synaptic junction; activation of the climbing fibers by a US causes a large complex set of activation spikes in the Purkinje cells.

inferior olive

Located in the brainstem, a nucleus of cells that conveys information about aversive stimuli, such as an airpuff US, up to both the interpositus nucleus and the cerebellar cortex.

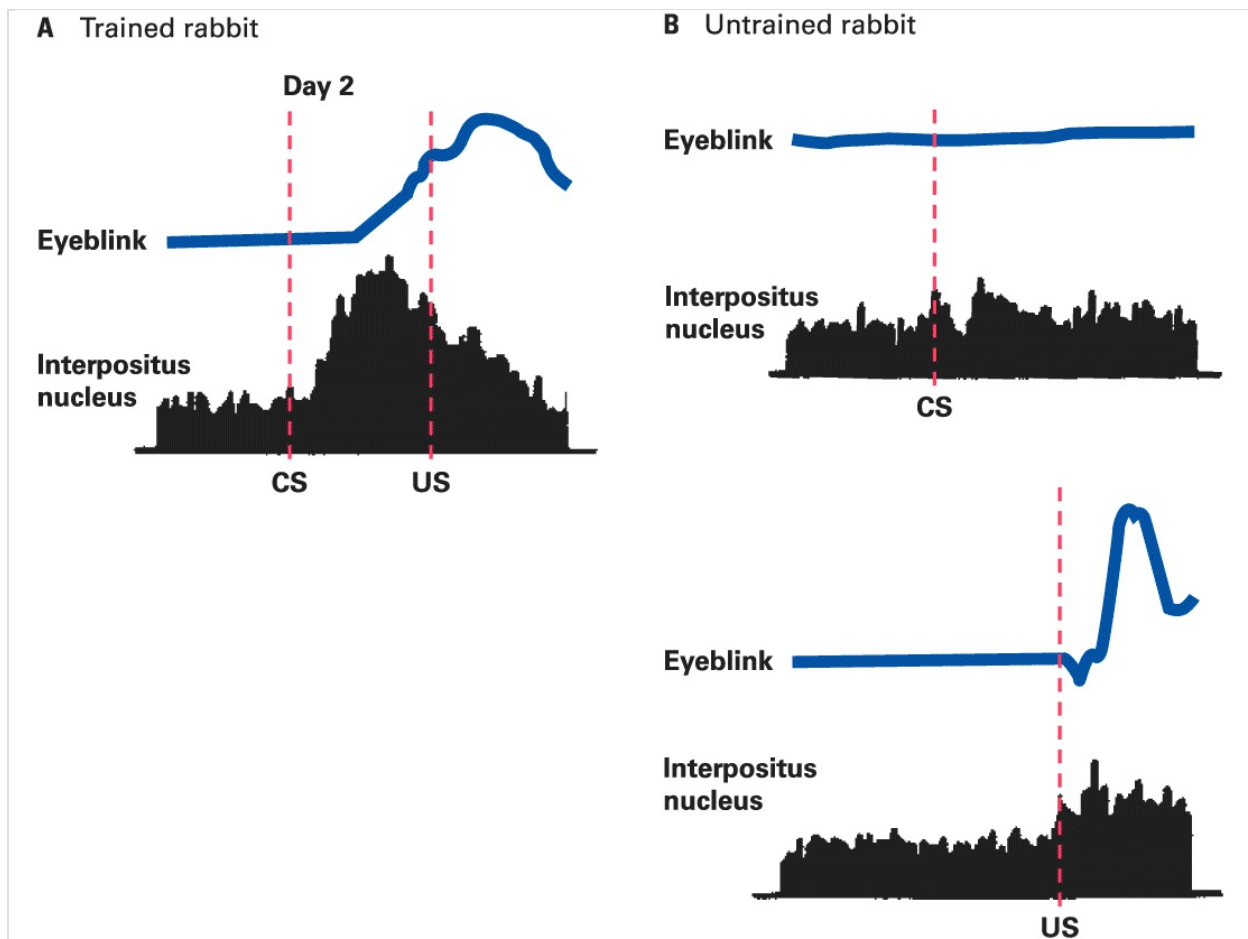
Complementing these two converging input pathways is a single output pathway for the CR, shown in red, which starts from the Purkinje cells. The Purkinje cells project down from the cerebellar cortex into the deep nuclei, where they form an inhibitory synapse (shown as a red rectangle) with the interpositus nucleus. To produce an eyeblink response, output from neurons in the interpositus nucleus travels (via several other, intermediary cells) to the muscles in the eye to generate the eyeblink CR. You may notice that [Figure 4.17](#) also includes an inhibitory pathway from the interpositus to the inferior olive, but we will postpone discussion of this pathway until later in the chapter. The UR pathway is not shown in [Figure 4.17](#) because that is an innate response; it is not learned and does not originate in, or require, the cerebellum. Instead, it is a reflex circuit, similar in principle to the spinal reflexes described in [Chapter 2](#).

The most important thing to note about this motor-reflex conditioning circuit (as diagrammed in

[Figure 4.17](#)) is that there are two sites in the cerebellum where CS and US information converge and, thus, where information about the CS–US association might be stored: (1) the Purkinje cells in the cerebellar cortex and (2) the interpositus nucleus. These two sites of convergence are intimately interconnected in the output pathway: the Purkinje cells project down to the interpositus nucleus with strong inhibitory synapses.

Electrophysiological Recording in the Cerebellum

When an electrode is inserted into the interpositus nucleus (one of the two sites where CS and US information converge, and the final exit point of CR information from the cerebellum), the recordings of spiking neurons during conditioned eyeblink responses display a pattern that corresponds very closely to the pattern of the eyeblinks themselves, as seen in [Figure 4.18A](#), taken from a rabbit after 1 day of tone CS–US training ([McCormick & Thompson, 1984](#)). The main difference between the two patterns is that the neural activity occurs just a few milliseconds before the actual behavior. The upper blue line shows the eyeblink behavior (the extent of eyelid closure over time), while the lower graph shows the frequency of neuron firing in the interpositus nucleus, averaged over several rabbits and several trials.



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FIGURE 4.18 Electrophysiological recordings in the rabbit cerebellum during classical conditioning (A) Response of a trained rabbit to the CS. (B) Response of an untrained, naive rabbit to the CS alone (top) and to the US alone (bottom). The blue lines show the eyeblink behavior (the extent of eyelid closure over time), while the graphs beneath them show the frequency of neuronal firing in the interpositus nucleus.

Data from [McCormick & Thompson, 1984](#).

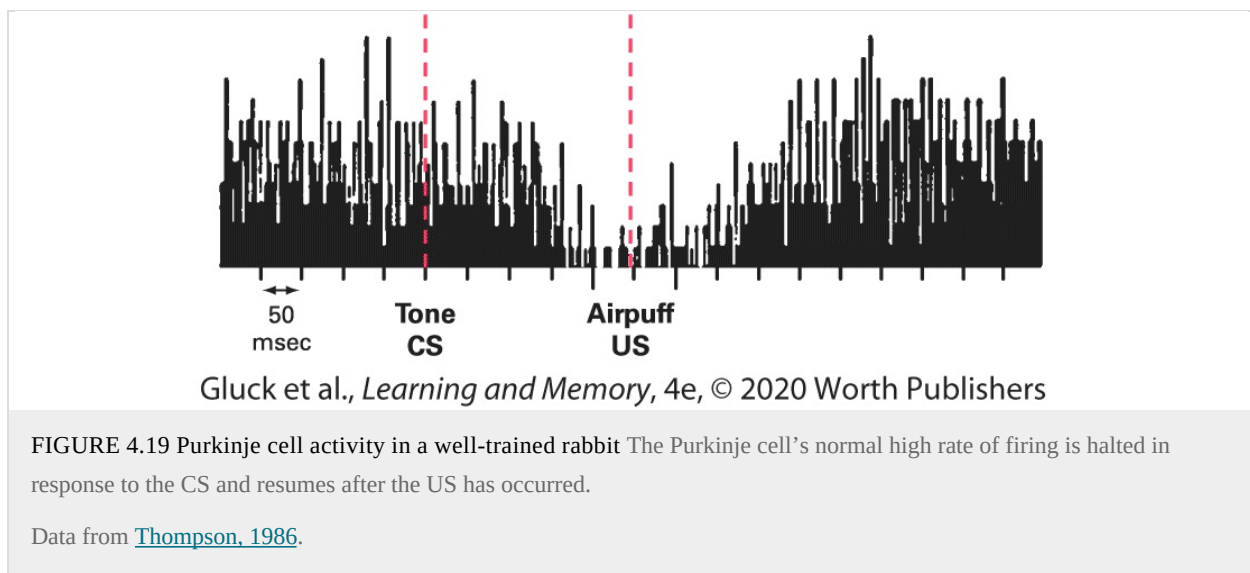
Description

The first illustration labeled A shows electrophysiological recordings in a trained rabbit. The graph line shows the eyeblink response of a trained rabbit, after the C S and before the U S. The graph of the firing of the interpositus nucleus shows a spike in the firing rate after the C S and before the U S.

The second illustration labeled B shows electrophysiological recordings in a trained rabbit. When C S is introduced to an untrained rabbit, there is no eyeblink response either on the graph line or in the firing of the interpositus nucleus. When U S is introduced to the untrained rabbit, the graph line spikes in response and the firing rate of the interpositus nucleus increases.

Researchers have also recorded unpaired, CS- or US-alone trials in naive rabbits. In both cases, on trials where there is no CR (eyeblink), there is no activity in the interpositus nucleus, as shown in [Figure 4.18B](#). The lack of substantial interpositus activity in a US-alone trial (despite a strong eyeblink UR) confirms that the cerebellum is responsible for conditioned eyeblink CRs only and not for the unconditioned eyeblink URs.

[Figure 4.19](#) shows the firing rates recorded for a single Purkinje cell in a well-trained rabbit, with the time of the CS onset and the US indicated beneath. Purkinje cells spontaneously fire all the time, even when nothing is happening. However, in a well-trained animal, many of these cells *decrease* their firing in response to the tone CS, as shown in [Figure 4.19](#). What causes this decrease in Purkinje cells' firing in response to the CS? Researchers believe that the repeated pairing of a CS with the US-induced firing of the Purkinje cells causes a decrease, or depression, in the strength of the parallel fiber synapses on the Purkinje cells. This depression of synaptic efficacy results in a depression of CS-related activity in the Purkinje cells in a well-trained animal that has experienced many CS–US pairings. The decrease, called *long-term depression* (LTD), is the opposite of the more common *long-term potentiation* (LTP), or increase in synaptic efficacy, described in [Chapter 2](#) as the basis for Hebbian learning between two neurons.



What is the downstream functional consequence of the Purkinje cells being turned off in response to a CS? Looking back at the diagram of cerebellar circuitry in [Figure 4.17](#), note that Purkinje cells *inhibit* the interpositus nucleus, the major output pathway driving the conditioned

motor response. Shutting off the Purkinje cells removes inhibition from the interpositus, freeing the interpositus to fire (as in [Figure 4.18A](#)).

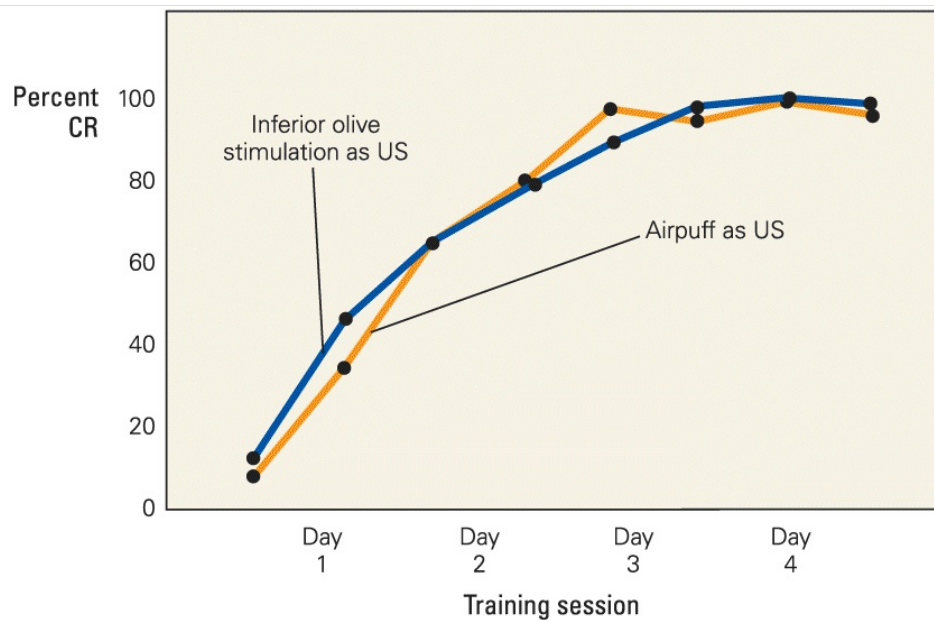
Brain Stimulation as a Substitute for Behavioral Training

What if we knew exactly which pathways in your brain would change as a result of reading the words on this page? If we knew this, we might be able to put electrodes in your brain and electrically stimulate those pathways in just the right pattern, at just the right time, to mimic the effect of reading this text. If that were possible, you wouldn't have to bother reading this book any further or studying for the final exam. Instead, you could stimulate a few neural pathways, create a little synaptic change, and then take the final exam and score an A+, even if you had never opened the textbook or sat through your professor's lectures! Science fiction, right?

Unfortunately, yes, because we do not yet know exactly where or in what way complex learning is stored in the brain. However, for simpler forms of learning, like eyeblink conditioning, this scenario is not only possible, it has been carried out. Through electrical brain stimulation of the CS and US pathways shown in [Figure 4.17](#), an experimenter can create conditioned eyeblink responses in the rabbit that are indistinguishable from those arising from behavioral training.

Recall that different parts of the pontine nuclei respond to different kinds of sensory input, such as auditory tones or visual signals, as illustrated in [Figure 4.17](#). It is even possible to find a specific region in the pontine nuclei that responds to a *particular* tone. As a result, it is possible to condition rabbits merely by pairing electrical stimulation of the pontine nuclei (the CS) with electrical stimulation of the inferior olive (the US) without presenting any external stimuli (airpuff or tone). After training with this type of brain stimulation, rabbits give precisely timed, reliable eyeblink responses the very first time they hear an actual tone corresponding to the pontine nuclear region that was stimulated, just as if they had been trained all along with tones and airpuffs ([Steinmetz, Lavond, & Thompson, 1989](#)).

In these studies, direct stimulation of the inferior olive causes the rabbit to blink; this stimulation, as stated above, can be substituted for an airpuff US. As shown in [Figure 4.20](#), similar conditioning over 4 days of training is seen whether an actual airpuff US (orange line) or a stimulation of the inferior olive (blue line) is used ([Steinmetz et al., 1989](#)).



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FIGURE 4.20 Substituting stimulation of the inferior olive for a US Four days of training using stimulation of the inferior olive as the US (blue line) produces the same amount of conditioned eyeblink response as 4 days of training with an airpuff US (orange line).

Data from [Steinmetz, Lavond, & Thompson, 1989](#).

Description

The X-axis shows Training session, with values ranging from Day 1 to Day 1, with an interval of 1 day. The Y-axis shows Percent C R, with values ranging from 0 to 100, at an increment of 20 units. The data in the graph is as follows:
Two line graphs, with nearly identical learning curves over four days of training for two methods: stimulating the inferior olive and applying the airpuff.

Thus, rabbits that have had their inferior olives and pontine nuclei electrically stimulated will “pass the eyeblink test” much as if they had gone through days of tone–airpuff training. As with the science fiction fantasy alluded to earlier, stimulating the correct pathways creates learning that seems indistinguishable from the conditioned response of a rabbit that has gone through the usual training with tones and airpuffs.

Impaired Conditioning Following Cerebellar Damage

Another experimental approach for investigating the neural bases of classical conditioning is to introduce brain lesions—that is, to selectively remove small areas of the brain—and observe the consequences. Recall that the interpositus nucleus (see [Figure 4.17](#)) projects information about

the CR out of the cerebellum. Thus, without the interpositus nucleus, you would expect that there could be no CR. This is exactly what Thompson and colleagues found: removing even 1 cubic millimeter of tissue from the interpositus nucleus completely and permanently abolished all previously learned conditioned responses and prevented all future eyeblink learning.

In contrast to lesions of the interpositus, which totally abolish learned eyeblink CRs, lesions of the cerebellar cortex (including the Purkinje cells) disrupt but do not eliminate eyeblink conditioning. Animals with lesions of the cerebellar cortex show small, poorly timed conditioned CRs ([Perret, Ruiz, & Mauk, 1993](#)). For other studies, researchers developed mutant mice with a genetic variation that causes selective degeneration of Purkinje cells. These mutant mice are slow at learning eyeblink conditioning, much like animals that have had their cerebellar cortices physically removed ([Chen, Bao, Lockard, Kim, & Thompson, 1996](#)). Together, these lesion and mutant studies provide strong converging evidence that the interpositus nucleus is involved in the formation and execution of the conditioned response, while the cerebellar cortex is involved in response timing.

Given the critical role of the cerebellum in motor-reflex conditioning, it is not surprising that patients with cerebellar damage display significant deficits in acquiring eyeblink conditioning. Such patients are slower to learn the CR and show low overall frequency and abnormal timing of CRs ([Daum et al., 1993](#)). Interestingly, patients who have undergone surgery that spares the deep nuclei are able to acquire a little conditioning, but patients with more extensive cerebellar damage show no conditioning at all. It is important to note that cerebellar damage does not impair *all* forms of associative learning, however. For example, cerebellar patients perform within the normal range on learning verbal associations, such as matching names with faces, which suggests that other areas of the brain play roles in these more abstract tasks ([Daum et al., 1993](#)). There is also a clear lateralization of cerebellar involvement in eyeblink conditioning: damage to the left cerebellum interferes only with conditioning to the left eye, whereas damage to the right cerebellum interferes only with conditioning to the right eye. This is true in both rabbits and humans ([Thompson & Krupa, 1994](#); [Woodruff-Pak & Lemieux, 2001](#)).

Genetics offers additional insights into human eyeblink conditioning. Irene Daum and colleagues have studied several groups of patients in whom chromosomal irregularities cause abnormalities and degeneration in either the cortical Purkinje cells or the deep nuclei ([Daum et al., 1993](#)). They

have found that patients with genetic abnormalities of the deep nuclei are severely impaired at acquiring the eyeblink CRs, while those with abnormalities in the Purkinje cells show more mixed results. These genetic studies provide additional evidence that the deep cerebellar nuclei are essential for learning the CR, whereas the Purkinje cells in the cerebellar cortex exert some modulating but nonessential influence on this learning.

Error Correction Through Inhibitory Feedback

As [Chapter 2](#) describes, long-term potentiation (LTP) of a synapse occurs when simultaneous activity in two adjoining neurons leads to a strengthening of the connecting synapse (remember: “neurons that fire together wire together”). LTP is a mechanism for synaptic change that occurs whenever two adjoining neurons fire at the same time; it is thus much simpler than the error-correcting rule of the Rescorla–Wagner model, in which associative changes depend on many inputs (such as all the CSs present on a trial). Given this disparity, the Rescorla–Wagner model of learning probably does not describe what takes place in a learning brain at the cellular level, but the error-correction mechanisms the model predicts do appear to emerge from brain circuits.

Look again at the cerebellar network in [Figure 4.17](#), and you will see an additional pathway within the cerebellum we have not yet discussed: an inhibitory feedback pathway from the interpositus nucleus to the inferior olive. In a well-trained animal, the production of a CR, through activation of the interpositus nucleus, will in turn inhibit the inferior olive from sending US information to the Purkinje cells in the cerebellar cortex ([Sears & Steinmetz, 1991](#)). This means that activity in the inferior olive will reflect the actual US (the airpuff in [Figure 4.17](#)) minus (due to inhibition) the expected US. (The expected US is determined by the interpositus activity, which is what drives the eyeblink CR.) Actual US minus expected US: sound familiar? It should. This is the same calculation the Rescorla–Wagner model uses to compute the prediction error in a trial, which is then used to determine how much weight should accrue to the CS association.

If the inferior olive is where the brain codes the prediction error during conditioning, then we should be able to predict changes in the firing of the inferior olive based on the Rescorla–Wagner model ([Gluck, Allen, Myers, & Thompson, 2001](#); [Gluck, Reifsnider, & Thompson, 1990](#)). During CS–US acquisition training, the prediction error diminishes on each successive learning trial, so we would expect to see inferior olive activity also diminish the more the US is predicted

by the trained CS. Eventually, when the CR is well learned, there should be very little activity in the inferior olive (that is, when error in the Rescorla–Wagner model is close to zero). What happens matches the predictions exactly: inferior olive activity starts off high early in training and then gradually diminishes as the conditioned response is acquired ([Sears & Steinmetz, 1991](#)).

This interpretation of how the cerebellar circuits compute the changes in associative weight called for in the Rescorla–Wagner model implies that Kamin’s blocking effect (the clearest experimental evidence for error-correction learning) should depend on the inhibitory pathway from the interpositus to the inferior olive. This prediction was confirmed in a study by Thompson and colleagues. The researchers first trained rabbits to give reliable eyeblink responses to a tone CS and then injected a drug into the interpositus that temporarily disabled the inhibitory connection from the interpositus to the inferior olive. With this pathway disabled, they predicted, the inferior olive’s activity would reflect the presence of the actual US and not the difference between this actual US and the expected US.

The rabbits were then given phase 2 blocking training, in which a compound tone-and-light CS was paired with the US. The rabbits showed high inferior olive activity whenever the US was presented, whether or not a conditioned response was generated. As a result, in phase 3, the rabbits gave a strong response to the light CS rather than the muted or absent response that would normally be seen in the blocking paradigm. In other words, by disabling that one inhibitory pathway, which is essential for computing prediction error, Thompson and colleagues were able to “*block blocking*” ([Kim, Krupa, & Thompson, 1998](#)). These and related results suggest that the cerebellar–inferior olive circuit plays a role in the execution of Rescorla and Wagner’s error-correction rule.

TEST YOUR KNOWLEDGE

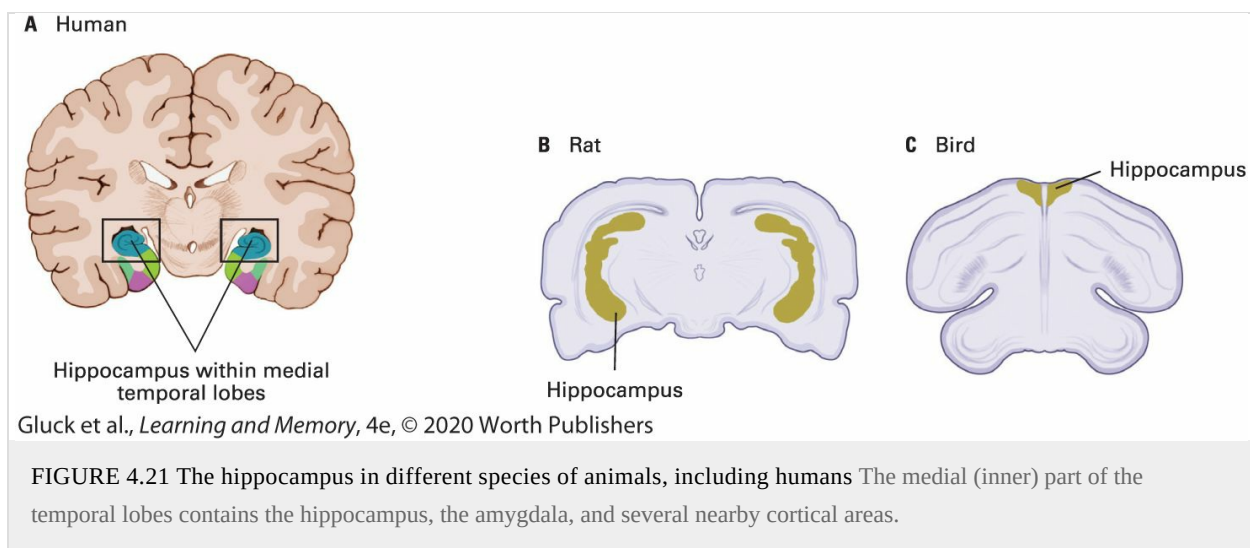
The Cerebellum in Motor-Reflex Conditioning

1. What is the role of the Purkinje cells in the cerebellar cortex? Discuss the evidence that suggests this.
2. What are the two main cerebellar regions and the major sensory-input pathways to the cerebellum? Where do these two pathways in the cerebellum converge?
3. How do electrophysiological recordings in the rabbit cerebellum during classical conditioning demonstrate that the cerebellum is responsible for conditioned responses and not for unconditioned responses?

([Answers appear at the end of the chapter.](#))

The Hippocampus in CS Modulation

Error correction, as explained by the Rescorla–Wagner model, is only one mechanism at work in classical conditioning. Another, CS modulation, was suggested by the attentional theory of Mackintosh, as noted earlier. This explanation invokes the hippocampus, which, as described in [Chapter 2](#), is a string-bean-shaped structure that, in humans, lies just inward from the ears ([Figure 4.21A](#)). Other mammals, such as rats, also have hippocampi; in mammals with small cortical area, the hippocampus is large relative to total brain size ([Figure 4.21B](#)). Other types of animals, such as birds, fish, and reptiles, also have brain areas that are thought to be analogous to the mammalian hippocampus ([Figure 4.21C](#)).



Description

The illustration labeled A shows a cross-sectional view of the human brain. The small structures on the inside of the temporal lobes are labeled, Hippocampus within medial temporal lobes.

The illustration labeled B shows a cross-sectional view is simple line drawing of a cross-sectional view of a rat's brain. The region forming large arcs on both sides of the brain is labeled, Hippocampus.

The illustration labeled C shows a cross-sectional view of a bird's brain. The small area at the tip of the brain is labeled, hippocampus.

The hippocampus is not necessary for learning new conditioned responses. For example, animals or humans with hippocampal damage are able to learn a basic conditioned eyeblink response quite normally. Nevertheless, electrophysiological recordings of animals show that the

hippocampus is very active during conditioning, especially early in training. What role does the hippocampus play in conditioning? One way to find possible clues to its role is to look at more complex conditioning paradigms, such as latent inhibition (described in [Table 4.7](#)). As you learned in [Section 4.1](#), latent inhibition is demonstrated when, before training, an organism is exposed to a cue unassociated with a US; later, during conditioning, the organism is then slow to learn that the cue does predict a US.

As you also learned, the Rescorla–Wagner model is *not* able to explain the phenomenon of latent inhibition. If the Rescorla–Wagner model’s error-correction process cannot explain latent inhibition and if the cerebellum implements the error-correction principle, then perhaps other brain regions involved in classical conditioning besides the cerebellum are responsible for latent inhibition. Might the hippocampus be such a region? If so, then the theories that capture classical conditioning phenomena other than error-correction learning might offer some ideas of how the hippocampus may contribute.

For example, the CS modulation theory discussed earlier in this chapter suggests that latent inhibition depends on the salience of sensory cues. Perhaps the hippocampus plays a role in determining stimulus salience. If so, then an animal *without* a hippocampus should *not* exhibit CS modulation effects such as latent inhibition. In fact, this is exactly what researchers have found: removing the hippocampus (and associated cortical input regions) eliminates the latent inhibition effect in classical conditioning of the rabbit eyeblink reflex ([Shohamy et al., 2000](#); [Solomon & Moore, 1975](#)).

In light of observations such as these, perhaps the Rescorla–Wagner model should be described as a model of the cerebellar contributions to motor-reflex conditioning in hippocampal-lesioned animals rather than as a model of conditioning in healthy, intact animals. It applies best to the brain regions responsible for error-correction learning, such as the cerebellum, but it does not explain the contributions of the hippocampus. Further discussion of the hippocampus—specifically, in the processing of sensory relationships while remembering new facts and events—appears in [Chapter 6](#), and [Chapter 9](#) describes some theories of how the hippocampus modulates sensory processing in various forms of learning and memory.

Invertebrates and the Cellular Basis of

Learning

[Chapter 3](#) introduces the sea hare *Aplysia* and its use by Eric Kandel and colleagues for studying the neural substrates of two simple forms of learning: habituation and sensitization. To briefly recap, *habituation* occurs when *Aplysia*'s siphon (see [Figure 3.9](#)) is repeatedly but lightly touched. Initially, the touches result in a gill-withdrawal reflex; however, each subsequent stimulation of the siphon elicits a progressively smaller response. The circuit for this learned response includes a sensory neuron (activated by touching the siphon) that makes an excitatory synapse with a motor neuron that controls the gill withdrawal (see [Figure 3.10](#)). The neural mechanism for habituation is thought to be a progressive decrease in the number of neurotransmitter (in this case, glutamate) vesicles available in the sensory neuron's axon for each successive stimulation of the siphon.

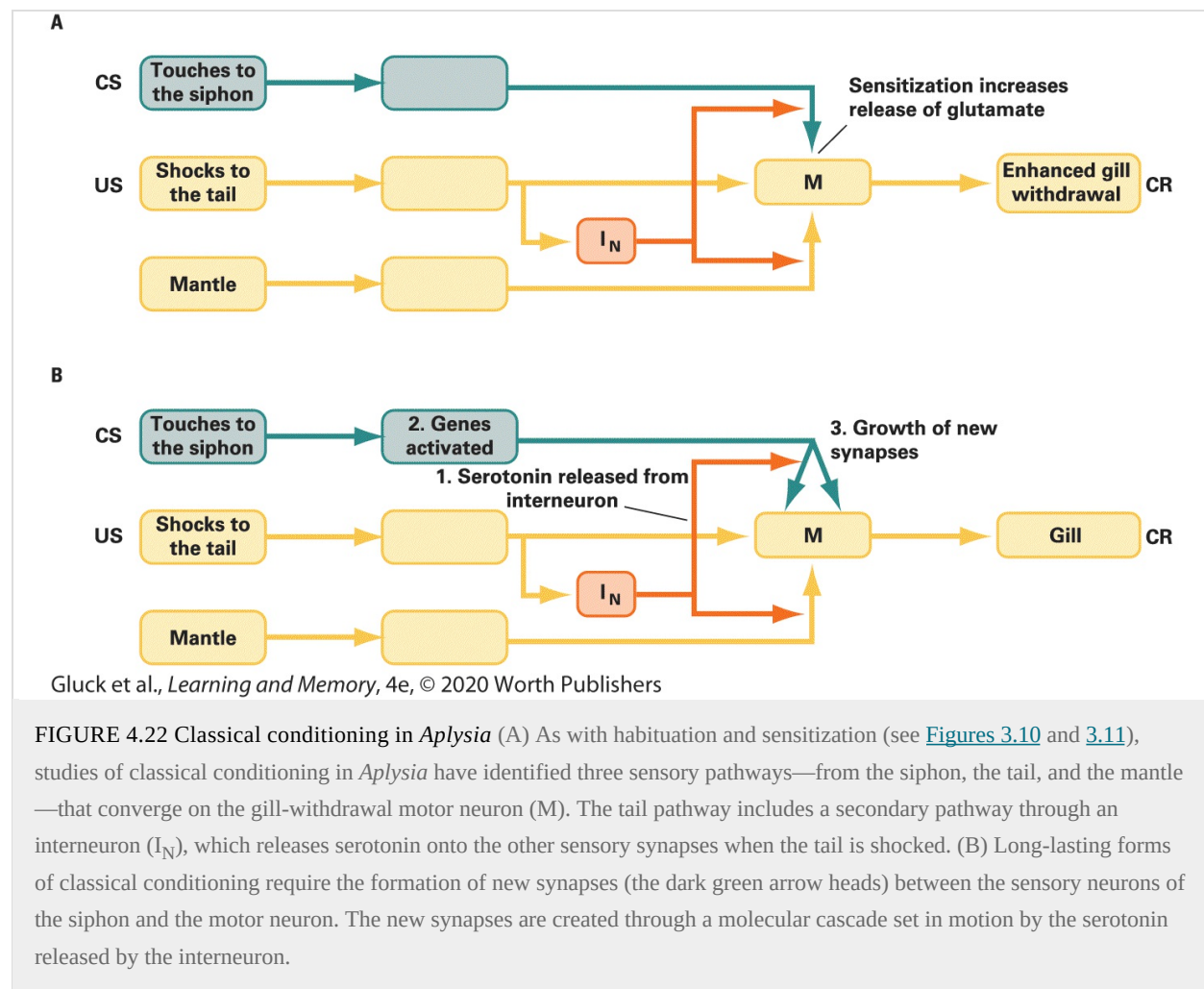
In contrast, *sensitization* is a global *increase* in responding to all or most stimuli following an unpleasant stimulus, such as an electric shock to *Aplysia*'s tail. The tail shock activates modulatory interneurons ("I_N" in [Figure 4.22](#)) that release serotonin onto the axon terminals of all the sensory neurons that project to the gill-withdrawal motor neuron. Serotonin increases the number of glutamate vesicles released when the sensory neuron is stimulated. This results in the generalized (non-stimulus-specific) increase in gill withdrawal elicited by all future stimuli, including touches on either the siphon or the mantle. The top two entries in [Table 4.9](#) summarize the key differences between these two forms of learning.

TABLE 4.9 Varieties of learning in *Aplysia*

Type of learning	Association formed?	Stimulus specific?	Mechanism(s)	Locus of effect
Habituation	No	Yes	Decrease in glutamate	Cellular process
Sensitization	No	No	Serotonin-induced increase in glutamate	Cellular process
Classical conditioning	Yes	Yes	1. Presynaptic activity-dependent enhancement of glutamate release from sensory neuron 2. Postsynaptic change in receptors of motor neuron 3. A cascade of intracellular molecular events that activate genes in the neuron's nucleus, causing an increase in the number of sensorimotor synapses.	Cellular process Structural change Structural change

What do you think would happen if both kinds of stimuli—touching the siphon and shocking the

tail—were repeatedly paired? Tom Carew, in collaboration with Kandel and other colleagues, showed that *Aplysia*'s gill-withdrawal reflex can be classically conditioned, as illustrated in [Figure 4.22A](#). After touches to the siphon (a potential CS) have been repeatedly paired with shocks to the tail (a US), subsequent touches to the siphon will elicit an enhanced gill-withdrawal response (the CR; [Carew, Hawkins, & Kandel, 1983](#)). The enhanced gill-withdrawal response to the siphon-touch CS following paired training is considerably greater than the generalized sensitization that occurs from presentation of the tail shock alone. Moreover, this classically conditioned gill-withdrawal CR is specific to touches to the siphon and does not generalize to other stimuli, such as a touch on the mantle (potentially another CS, as shown at the bottom of [Figures 4.22A](#) and [4.22B](#)).



Description

The illustration labeled A shows the habituation and sensitization pathway for *Aplysia*. The three sensory pathways are

as follows. Siphon, tail, and mantle. A text box reads, Touches to the siphon is connected to the motor neuron M on a C S pathway. The text box reads, Shock to the tail is connected to the motor neuron M on a U S pathway, which generates an interneuron I. The text box that reads, Mantle is connected to the motor neuron M by a Sensory neuron. A label beside the motor neuron reads, Sensitization increases release of glutamate. The motor neuron M is connected to the gill muscles and the text box reads, Enhanced gill withdrawal, in the C R pathway.

The illustration labeled B shows the habituation and sensitization pathway for *Aplysia* after the formation of new synapses. A text box that reads, Touches to the siphon, in the C S pathway leads to a text box that reads, Genes activated., which is further has a release on new synapse connected to the motor neuron. The text box reads, Shock to the tail is connected to the motor neuron M on a U S pathway, which generates an interneuron I. The text box that reads, Mantle is connected to the motor neuron M by a Sensory neuron. The motor neuron M is connected to the gill muscles and the text box reads, Enhanced gill withdrawal, in the C R pathway.

What happens inside the nervous system of *Aplysia* when these two stimuli are paired? Kandel and colleagues demonstrated that paired training produces an increase in the glutamate vesicles that are released in the siphon sensory neuron's synapse on the motor neuron, much like an exaggerated form of the mechanism for sensitization described in [Chapter 3 \(Hawkins, Abrams, Carew, & Kandel, 1983\)](#). This implies that a cellular mechanism for classical conditioning can be understood as an elaboration of the same cellular mechanism that leads to sensitization.

The enhancement of glutamate release in the sensory neuron synapse, brought on by the pairing of the siphon touch and tail shock, is called an **activity-dependent enhancement** because it depends on activation of the sensory neuron prior to the administration of the US. Earlier in this chapter, we discussed how classical conditioning of the rabbit eyeblink response is sensitive to the order and timing of the tone CS and the airpuff US. The same holds true for conditioning of *Aplysia*'s gill withdrawal: conditioning occurs only if the siphon-touch CS is presented about half a second before the tail-shock US. If the US occurs much later (more than 2 seconds after the CS) or before the CS, nothing other than nonspecific sensitization will occur. Thus, after sensory stimulation, whatever process occurs within the neuron to prime the neuron for an increase in glutamate release has a time course of about half a second.

activity-dependent enhancement

Paired training of CS and US that produces an increase in the glutamate vesicles released from sensory neurons to motor neurons.

To summarize, Kandel and colleagues demonstrated that activation of the sensory neuron in *Aplysia*'s siphon has at least three consequences. First, it causes the motor neuron to fire, by the release of the neurotransmitter glutamate into the synapse. Second, it causes a decrease in

glutamate vesicles available for any subsequent stimulation of the sensory neuron, resulting in habituation. Third, it primes the synapse through a series of intracellular events lasting about half a second, such that the subsequent presence there of the neurotransmitter serotonin (released following activation of an aversive tail shock) creates an increase in future glutamate release. In this way, the pairing of the sensory stimulus CS and the tail shock US results in a classically conditioned increase in gill withdrawal (CR).

Presynaptic Versus Postsynaptic Changes During Learning

The activity-dependent enhancement of the sensory neuron's release of glutamate onto the motor neuron in *Aplysia* is a presynaptic form of synaptic plasticity because, like sensitization, it involves a change in the sensory neuron. However, the story is actually more complicated. There is also a postsynaptic mechanism for this conditioning that involves changes in neurotransmitter receptors on the motor neuron ([Bao, Kandel, & Hawkins, 1998](#)). Thus, the mechanisms for classical conditioning in *Aplysia* include both presynaptic and postsynaptic changes in the circuits connecting the CS and the CR, as summarized in [Table 4.9](#).

One advantage of *Aplysia* as a model system for studying the intracellular molecular pathways of learning is that it is possible to identify key neurons (such as entire memory-trace circuits), remove them from the animals, and keep those neurons functioning in a culture dish. By isolating the key circuits for learning and studying them outside the animal, Kandel and colleagues were able to explore the question of how long-term changes in *Aplysia* circuitry could account for long-lasting forms of classical conditioning. The search for the answer to this question took neuroscientists back to the very origins of who we are—to our genes—and has given rise to an important new field: the molecular genetics of memory. (It also won Kandel the Nobel Prize for Physiology or Medicine in 2001.) As discussed in more detail in [Chapter 12](#), genes are stretches of DNA (deoxyribonucleic acid) molecules, found in the nucleus of every cell, that encode information needed to produce protein molecules. Our genes play a critical role throughout our lives, guiding the maintenance and continuing growth and development of our bodies and brains, including the changes that result in long-lasting forms of memory.

Long-Term Structural Changes and the Creation of New

Synapses

Using recent advances in molecular biology techniques, Kandel and colleagues were able to show that the serotonin released by *Aplysia*'s interneurons following a tail-shock US does more than cause a short-term increase in the sensory neuron's release of glutamate; it also launches a cascade of intracellular molecular events that set the stage for long-term structural changes in the neuron. Following multiple pairings of the CS and US, protein molecules in the sensory neuron's synapse travel back up the axon of the sensory neuron, all the way to the cell body. There they switch on genes inside the nucleus of the neuron that in turn set in motion the growth of new synapses ([Figure 4.22B](#)).

More recent work by Kandel and others has identified two proteins that are found inside neurons and that play critical regulatory roles in this synapse-creation process. The first protein, CREB-1, activates genes in the neuron's nucleus that initiate the growth of new synapses. The second protein, CREB-2, plays an opponent role, inhibiting the actions of CREB-1. The creation of new synapses during learning requires a cascade of processes inside the cell that activate CREB-1 and suppress CREB-2.

What do you think would happen if functioning of the CREB-1 protein were impaired? Kandel and colleagues demonstrated that if CREB-1 is rendered inactive by injection of molecules into the neuron that compete with CREB-1's ability to activate genes for new synapses, the circuits subsequently fail to show long-lasting forms of associative learning ([Dash, Hochner, & Kandel, 1990](#)). Most important, the researchers also demonstrated that inactivation of CREB-1 does not affect the short-lasting forms of learning that depend only on increased glutamate release. This study provided critical evidence of a dissociation between short-lasting forms of learning, which do not require the CREB-1 protein, and long-lasting forms, which do.

In a related study, Kandel and colleagues showed that removing the influence of the opponent protein, CREB-2, had the opposite of the effect of inactivating CREB-1: with CREB-2 inactivated, long-lasting learning occurs rapidly at the sensory neurons, after even a single exposure to serotonin ([Bartsch et al., 1995](#)). The role of CREB molecules in modulating long-lasting forms of memory is not limited to *Aplysia*; increasing CREB-1 in fruit flies (*Drosophila*) allows them to learn much more rapidly than they usually do, while increasing their CREB-2 blocks the formation of long-term memories ([Yin et al., 1994](#)). The CREB molecules also play a

critical role in mammals' learning; studies in mice have shown that activity of CREB-1 in the hippocampus is critical to long-lasting, but not to short-term, increases in neuron-to-neuron associations arising from LTP ([Bourtchuladze et al., 1994](#)).

Studies of classical conditioning in *Aplysia* have demonstrated that anatomical changes in neural circuits, including the growth or deletion of synapses, are characteristic of long-lasting forms of memory. In contrast, short-term, labile forms of memory are associated with temporary intracellular changes within existing anatomical pathways, including shifts in the location, size, or number of neurotransmitter vesicles, which alter synaptic transmission efficacy. Thus, as discussed in [Chapter 3](#), the transition from short-term to long-term learning may be characterized as a shift from transmission-process-based changes within the neuron to structural changes within the neural circuits (see [Table 4.9](#)).

Interim Summary

- There are two sites in the cerebellum where CS and US information converges and that might therefore be locations for the storage of the CS–US association: (1) the Purkinje cells in the cerebellar cortex and (2) the interpositus nucleus. The interpositus nucleus lies along the only output pathway from the cerebellum; it forms part of the route by which the learned response travels to the motor systems that control behavior, such as an eyeblink CR.
- The inferior olive is believed to compute the degree to which a US is unexpected, providing the information necessary to implement Rescorla and Wagner's principle of error-correction learning in the cerebellum.
- The hippocampus participates in some forms of CS modulation in conditioning. This is consistent with data showing that an animal without a hippocampus does not exhibit CS modulation effects such as latent inhibition.
- Kandel and colleagues demonstrated that activation of the sensory neuron in *Aplysia*'s siphon by an external stimulation (such as presentation of a CS) primes the synapse, through a series of intracellular events lasting about half a second, such that the subsequent presence of serotonin (released following a US) creates an increase in future glutamate release. This results in a classically conditioned increase in gill withdrawal (CR) when the CS is next presented.
- After multiple pairings of the CS and US in *Aplysia*, protein molecules in the sensory neuron's synapse travel to the cell body, where they activate genes inside the nucleus of the neuron that trigger the growth of new synapses.

4.3 Clinical Perspectives

In this final section of the chapter, we focus on two clinical applications of classical conditioning. The first takes advantage of the ways in which drug addiction and drug abuse are intimately linked to classical conditioning. The other harnesses classical conditioning to reduce the amount of medication needed for treating a chronic disease.

Tolerance to Addictive Drugs

The role of learning and memory in drug addiction is a fascinating topic that we consider from several viewpoints in this textbook. [Chapter 5](#) explores the neural mechanisms of reward that are impaired by most drugs of abuse; [Chapter 8](#) discusses the role of the frontal lobes in inhibiting inappropriate behaviors and looks at how this role is compromised in drug addicts. The mechanisms of classical conditioning also play an important role in the cycle of addiction.

Early in this chapter, we discussed how automatic compensatory responses occur in body systems that have a mechanism for homeostasis, the tendency of the body (including the brain) to gravitate toward a state of equilibrium or balance. An addict's tolerance to drugs of abuse such as alcohol, cocaine, or ecstasy develops in the same way. As the addict's body adjusts to the drug effects (learning to anticipate the forthcoming "high"), larger and larger doses are required to produce the same high the addict experienced on first taking the drug. One way this happens is through conditioning: environmental cues that accompany drug use can classically condition the user to expect to receive the drug. In other words, the environmental cues (people, places, and so on) act like CSs associated with the drug (the US). The intense craving an addict feels in response to these cues is the CR and results from the body's conditioned compensatory response of lowering the levels of the brain chemicals enhanced by the drug in anticipation of the drug's arrival (more on the role of conditioning in drug addiction is presented in [Chapter 5](#)).

A potential by-product of such conditioned tolerance is that victims of heroin overdose are rarely novice users ([Siegel, 2001](#)). Rather, they tend to be longtime heroin addicts who have developed a high degree of tolerance to the drug but make the mistake of taking their usual dose in an unusual setting. For example, the situational cues that result in conditioned drug tolerance can include the room in which the drug is usually taken. You may recall reports of rock stars and others dying of heroin overdoses in hotel bathrooms, which were most likely far different from

the settings in which they were used to taking the drug. What might have happened is that they overdosed on what was otherwise their normal dosage of drug because, in this novel setting, their body was not prepared for the large influx of drug that it received.

Different methods of drug injection are another form of environmental cue that can become associated with drug expectation. One longtime heroin addict is reported to have died of an overdose when, looking for an accessible blood vein, he injected himself in his penis for the first time ([Winek, Wahaba, & Rozin, 1999](#)). Without the compensatory response to customary cues, the net effect of his usual dose was far greater than he was used to and resulted in his death (for reports of similar real-life cases, see [Siegel, 2001](#); [Siegel & Ellsworth, 1986](#)).

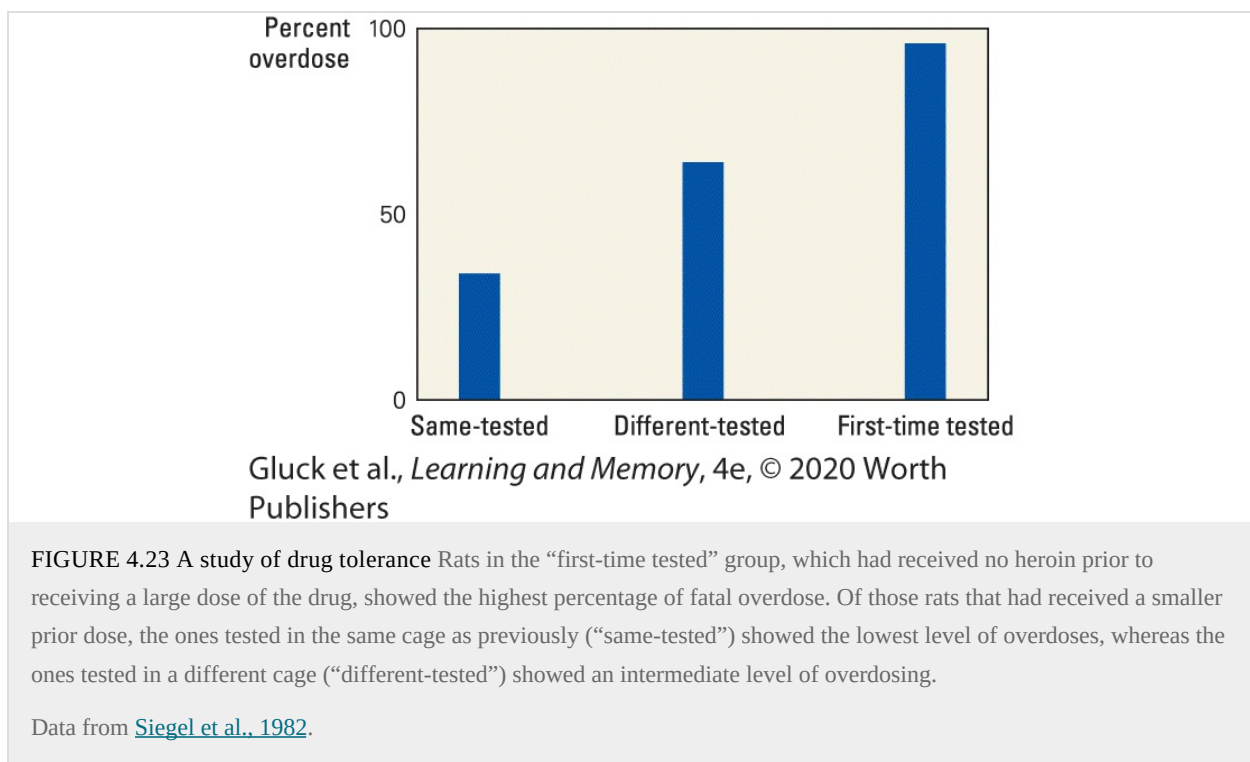
Even an unusual taste to a beverage can serve as a novel situational cue influencing the effects of drugs on the brain. This was demonstrated in a study in which college students showed greater cognitive and motor impairments when they consumed a given amount of alcohol in an unusual drink (in this case, a blue peppermint-flavored beverage) than when they had the same amount of alcohol in a familiar drink, such as beer ([Remington, Roberts, & Glauthier, 1977](#)). Perhaps this is yet another reason people get wilder at holiday parties, when they are drinking alcohol in sweet and bubbly holiday punches.



John Howard/DigitalVision/Getty Images

Would these students be more likely or less likely to get drunk compared to someone drinking straight whiskey?

Research has demonstrated conditioned tolerance in a wide variety of animal species. For example, Shepard Siegel and colleagues examined the effect of cues when heroin is administered to rats ([Siegel, Hinson, Krank, & McCully, 1982](#)). Siegel gave three groups of rats a fairly large (for their body weight) dose of heroin. The first group of rats had previously received a lower dose of heroin administered in the same cage and room where they were later tested with the large dose (the “same-tested” group in [Figure 4.23](#)). The second group of rats had also previously received the lower dose of heroin—but in a different cage and room (the “different-tested” group). Finally, the “first-time tested” group of rats were receiving heroin for the first time.



Description

The X-axis of the graph marks Percent overdose, and Y-axis marks three different groups such as Same-Tested, Different-Tested, First-Time. The data in the graph is as follows. The first-time tested group received a large dose of the drug, nearly 97 percent. the different- tested showed an intermediate level of overdosing, nearly 60 percent. the same-tested group shows the lowest level of overdoses, nearly 30 percent. All the data is approximate.

As shown in [Figure 4.23](#), Siegel and colleagues found that the large dose of heroin almost always (96% of the time) resulted in a fatal overdose in the “first-time tested” rats. In contrast, the

“different-tested” group showed some evidence of tolerance; only 64% of these rats suffered fatal overdose. But the “same-tested” rats, who were tested in the same environment in which they had previously been administered a lower dose of heroin, had a mortality rate of only 32%. Thus, these rats were protected from overdose by the conditioned tolerance they had learned during the administration of a lower dose of heroin in the same setting.

If drug tolerance is a form of classical conditioning, you might expect that the same rules and principles would apply to it as to eyeblink and other forms of conditioning. This appears to be the case. Recall the paradigm of latent inhibition discussed earlier and shown in [Table 4.7](#): pre-exposure to a CS delays subsequent learning of a CS–US association. If this latent inhibition effect applies to drug tolerance, then pre-exposing an animal to the cues that predict drug delivery should slow down development of learned tolerance to the drug. To test latent inhibition in the context of drug use, researchers have given animals an injection of an inert placebo to pre-expose them to the contextual cues (the sound and feel of getting an injection) of drug use. This pre-exposure does prove to slow down, by latent inhibition, the development of a cue–drug association where the cue is the CS and the drug is the US. In fact, this is exactly what Siegel and colleagues found in studies of morphine tolerance in rats ([Siegel, 1983](#)).

Such results provide compelling evidence for the applicability of Pavlovian analyses to learned drug tolerance. They suggest that drug tolerance and the loss of drug tolerance in novel drug-taking environments are mediated by basic processes of classical Pavlovian conditioning.

As discussed in [Section 4.1](#), an extinction paradigm should eliminate (or at least reduce) the association between conditioned cues and conditioned response when the cues are repeatedly presented alone and not paired with the prior US. If drug craving is viewed as a conditioned response, then the same principle of extinction should also apply—and indeed it does. In carefully controlled laboratory studies, rats that became addicted to alcohol showed significant extinction of this addiction through repeated nonreinforced exposure to experimentally manipulated cues that had previously been paired with administration of alcohol ([Krank & Wall, 1990](#)). In real life, however, it is very hard to both identify and extinguish all the cues that have become associated with drug use.

By deepening scientists’ understanding of the role of classical conditioning in drug addiction,

these and other studies may eventually provide new tools to help drug addicts overcome addiction. For example, perhaps further research on the extinction of conditioned responses will shed light on why addicts so often relapse when they are trying to kick their drug habit. You can read more about this issue in [“Learning and Memory in Everyday Life”](#).

LEARNING AND MEMORY IN EVERYDAY LIFE

Extinguishing a Drug Habit

Even in a well-controlled laboratory, extinguishing a cue and keeping it extinguished turns out to be extremely difficult. Mark Bouton and colleagues, studying the extinction of conditioned responses in rats, demonstrated that manipulations such as changing the context of the CS or just waiting for considerable time to pass (such as days or weeks) between one training session and the next training session often result in the extinguished cues reacquiring their conditioned associative properties ([Bouton, 2000](#)). Is it any surprise, then, that efforts to adapt extinction methods to therapy for drug addiction have yielded only mixed results at best ([Carroll, 1999](#); [Siegel & Ramos, 2002](#))? For example, many addicted individuals, after a long period of successful abstinence, can suffer relapse after a single drink or smoke. Bouton’s work suggests three principles that can help guide anyone trying to extinguish a habit or an association:

1. Because extinction effects are highly context sensitive, cue-exposure therapy should be conducted in as many different contexts as possible, including those that are part of the patient’s everyday life. This will prevent the extinction of drug craving from becoming dependent on any one context (such as a drug rehab center).
2. The extinction training should be spread out over time, rather than conducted all at once, because time (time of day, day of the week, month of the year ...) serves as a powerful context. A 2-week stint in a rehab clinic may not be enough to make a long-term difference. Multiple therapy sessions at different times and in different contexts would be more effective.
3. Whenever possible, the cue-exposure therapy should take place in the same contexts in which the original drug habits were acquired. Thus, it is better to go through the cue-exposure therapy at home than in a very unfamiliar setting, such as a drug rehabilitation center.

Reducing the Need for Medication or Reducing Its Side Effects

Classical conditioning in which a neutral stimulus is paired with a powerful medication is showing promise as a way of reducing the needed doses of medications that have unfortunate side effects. In one example, an 11-year-old girl suffering from the autoimmune disease lupus was able to benefit from this approach ([Olness & Ader, 1992](#); [Giang et al., 1996](#)). The standard treatment for lupus requires suppressing a person’s immune system in order to protect the tissues and organs from being targeted by the person’s own white blood cells. The chemotherapeutic drug the girl would have had to take orally, however, had many severe and disabling side effects.

To minimize her need to take the drug for an extended time, her doctors used classical conditioning methods in which she was initially given the drug in a liquid that tasted of cod liver oil and smelled of roses. Following that initial treatment, the doctors continued to give her the same rose-smelling cod liver oil every month, but they included the chemotherapeutic drug in the mixture only every other month. By cutting her total medication by half, they significantly reduced her suffering from side effects of the drug. At the same time, her body appeared to learn to suppress its own immune system in response to the compound cue of cod liver taste and rose smell. After several years of this treatment, she was still in remission from the disease.

Classical conditioning can also affect the occurrence and severity of side effects of medications. Many cancer patients undergo chemotherapy that causes severe nausea. Sometimes, too, if they eat a particular food around the same time as the chemo-induced nausea, they inadvertently develop a classically conditioned aversion to a food they have enjoyed in the past ([Jacobsen et al., 1993](#)). Other stimuli associated with the treatment can also become CSs that lead to nausea, such as the smell of the hospital room or the sight of the nurse. These consequences can be prevented or reduced, however, using lessons from classical conditioning. For instance, if a unique and novel food (such as an unusual flavor of ice cream) is paired with the chemotherapy, it may act as a “scapegoat” conditioned stimulus that blocks taste-aversion conditioning from accruing to other, normal foods the patient might encounter around the time of the chemotherapy treatment ([Bernstein, 1985](#)).

Interim Summary

- The situational cues that result in conditioned drug tolerance can be any sensory cues associated with drug use, including the feel of the needle and the method of injection.
- Rats are protected from heroin overdose by the conditioned tolerance they learned during the administration of lower doses in the same setting.
- Addiction can be partially reduced through Pavlovian extinction: rats that became addicted to alcohol showed significant extinction of this addiction through repeated nonreinforced exposure to experimentally manipulated cues that had previously been paired with administration of alcohol.
- Classical conditioning methods can be used to train people to suppress their own immune responses by pairing an immune-suppressing drug with a previously neutral odor and taste.

Synthesis

Classical conditioning is far more than just another behavioral process or tool for investigating brain systems: it is the mother of all memory systems. Evidence of classical “Pavlovian” conditioning surrounds us every day. Like Moira, who runs to the curb when she hears the musical jingle of an ice cream truck, and like Mandy, who has sworn off snails after a bout of illness, all the rest of us have been conditioned by cues in the environment to predict what might follow next.

In recent years, classical conditioning experiments have moved to the forefront of research into the physiological bases of learning because they afford exquisite control over what stimuli are presented as cues and because highly refined behavioral analyses and models have been developed as a result. Building on these analyses and models, neurobiological research has shown how different forms of classical conditioning are mediated by different brain systems, leading to fundamental insights into the neurobiology of learning and often providing tools that help us understand various clinical brain disorders.

Thus, influenced and guided by the error-correcting model of Rescorla and Wagner, along with other elegant mathematical theories, researchers are uncovering the neural bases of conditioning in a broad range of brain systems (including the cerebellum and the hippocampus), as well as the cellular basis of learning (including modifications at the level of the synapse). Links between conditioning and other forms of cognition, such as category learning, help us see how studies of neural mechanisms for learning can provide insights into the behavioral and neural bases of human cognition.

Meanwhile, the studies in invertebrates illustrate the general biological principle that evolution does not work like an engineer, creating new specialized systems for each new function. Instead, evolution works like a tinkerer, using preexisting components (seen in *Aplysia*, for example) in slightly modified form to perform new functions (in rats and other mammals), such as learning to predict aversive outcomes or categorize patients into one or another disease category. The behavioral and biological processes for classical conditioning are the basic building blocks, the biological alphabet, from which more complex forms of learning emerge in all species, including humans.

Chapter 4 Review

Know Your Key Terms

[activity-dependent enhancement](#)

[appetitive conditioning](#)

[associative weight](#)

[aversive conditioning](#)

[blocking](#)

[cerebellum](#)

[classical \(Pavlovian\) conditioning](#)

[compound conditioning](#)

[conditioned response \(CR\)](#)

[conditioned stimulus \(CS\)](#)

[conditioned taste aversion](#)

[CS modulation theory](#)

[delay conditioning](#)

[error-correction learning](#)

[extinction](#)

[eyeblink conditioning](#)

[homeostasis](#)

[inferior olive](#)

[interpositus nucleus](#)

[interstimulus interval \(ISI\)](#)

[latent inhibition](#)

[overshadowing](#)

[prediction error](#)

[Purkinje cell](#)

[tolerance](#)

[trace conditioning](#)

[trial-level model](#)

[unconditioned response \(UR\)](#)

[unconditioned stimulus \(US\)](#)

[US modulation theory](#)

Quiz Yourself

1. The relationship between a US and a UR does/does not involve learning. ([p. 123](#))
2. In eyeblink conditioning, the blink is both a(n) _____ and a(n) _____, although they differ in their _____. ([pp. 129–130](#))
3. In most conditioning paradigms, extinction is faster/slower than the original acquisition of the conditioned response. ([p. 133](#))
4. _____ conditioned responses in the body are most often the result of a biological mechanism called _____. ([p. 132](#))
5. Evidence that extinction is more than just unlearning comes primarily from studies that look at shifts in _____ between learning and testing. ([pp. 133–134](#))
6. When two cues compete to predict a US or other outcome, the one that is most strongly learned is usually the cue that is learned _____, as revealed in studies of blocking. ([p. 136](#))
7. Latent inhibition cannot be explained by the Rescorla–Wagner model because during pre-exposure, there is no _____. ([p. 147](#))
8. Beneath the _____ cells of the cerebellar cortex lie the cerebellar deep nuclei, including the _____ nucleus. ([p. 154](#))
9. CS information travels up to the deep nuclei of the cerebellum along axon tracts called the _____. ([p. 154](#))
10. An airpuff US to the eye activates neurons in the _____, a structure in the lower part of the brainstem. ([p. 155](#))
11. Purkinje cells inhibit/excite the interpositus nucleus, the major output pathway driving the conditioned motor response. ([p. 155](#))
12. Animals with lesions to the cerebellum show CRs, but they are _____. ([p. 158](#))

13. Latent inhibition and other expressions of CS modulation are impaired or eliminated by lesions to the _____. (p. 161)
14. The neural mechanism for habituation is thought to be a progressive decrease in the number of vesicles containing the neurotransmitter _____, available in the sensory neuron's axon. (p. 161)
15. The _____ of the sensory neuron's release of glutamate onto the motor neuron is a presynaptic form of _____. (p. 163)
16. Two proteins found inside neurons play critical regulatory roles in the synapse-creation process. The first protein, CREB-1, activates genes in the neuron's nucleus that _____ the growth of new synapses. The second protein, CREB-2, _____ the actions of CREB-1. (p. 164)
17. Rats can be protected from overdose by the _____ they acquired through the administration of lower doses of heroin in the same setting. (p. 167)
18. Appealing because of its simplicity, the _____ model has served as a starting point for many promising models of learning. (p. 143)
19. The learning that takes place in order to avoid or minimize the consequences of expected aversive events is known as _____. (p. 126)

Concept Check

1. Returning to our stock analysts Doris and Herman, consider what would happen if Doris showed up every day to work but Herman came in only every now and then. On the days that Doris works alone, she does a great job of predicting the stock market. But on the days that Herman shows up, the pair of them do a lousy job. What would you think about Herman? You'd probably conclude that he is no great asset. In fact, worse than being a do-nothing, he seems to interfere with Doris's ability to perform. Maybe he distracts her by talking about tennis when she should be working. You might say that Herman inhibits Doris's predictive value. This is similar to what occurs in conditioning procedures in which a tone cue is always followed by the US, except

when the tone appears as part of a compound tone-and-light stimulus. On these compound trials, no US occurs. What does the Rescorla–Wagner model predict will happen to the associations during this training?

2. A recovering drug addict attends therapy sessions in which cue-exposure therapy is used. The addict is exposed to drug-related stimuli (e.g., photos of common drug-taking environments, drug paraphernalia) in the therapy center several times a week for an extended period of time. Why might this treatment fail?

Answers to Test Your Knowledge

Pavlov's Experiment

CS = dog drool, CR = write in book

Classical Conditioning in Everyday Life

1. US, female model; UR, sexual arousal (most likely in men, who are the targeted customers); CS, the car; CR, arousal of pleasurable feelings at the thought and sight of the sports car; appetitive conditioning
2. US, hot pizza for dinner; UR, anticipation of dinner and salivation; CS, smell of cardboard burning; CR, hunger pangs and salivation; appetitive conditioning

Contrasting the Rescorla–Wagner and Mackintosh Models

1. US, CS
2. (a) Mackintosh; (b) Rescorla–Wagner

The Cerebellum in Motor-Reflex Conditioning

1. The Purkinje cells are involved in response timing. The evidence includes findings that lesions of the cerebellar cortex lead to smaller and poorly timed CRs and that mice with degeneration of Purkinje cells are slow to learn eyeblink conditioning.

2. The two main cerebellar regions are the cerebellar cortex containing Purkinje cells and the cerebellar deep nuclei. The major sensory-input pathways to the cerebellum are the CS input pathway and the US input pathway. The two sites of convergence are the Purkinje cells and the interpositus nucleus.
3. Researchers recording unpaired, CS- or US-alone trials in naive rabbits found that where there is no CR, there is no activity in the interpositus nucleus. Despite a strong UR (eyeblick) during the US-alone trials, there was a lack of substantial interpositus activity.



Chapter 5 Operant Conditioning

Learning the Outcome of Behaviors



Jean-François Ducasse/imageBROKER/Alamy

Behavioral Processes

The “Discovery” of Operant Conditioning

Learning About Stimuli and Responses

Learning About Outcomes

Learning and Memory in Everyday Life: Drug-Detecting Dogs

Learning and Memory in Everyday Life: The Problem with Punishment

Putting It All Together: Building the $S^D \rightarrow R \rightarrow O$ Association

Choice Behavior

Brain Substrates

The Dorsal Striatum and Stimulus–Response ($S^D \rightarrow R$) Learning

The Orbitofrontal Cortex and Learning to Predict Outcomes

Mechanisms of Reinforcement Signaling in the Brain

Punishment Signaling in the Brain

Clinical Perspectives

Drug Addiction

Behavioral Addiction

Treatments for Addiction

FROM 1977 TO 1992, the country of Mozambique was caught in a brutal civil war. When the conflict ended, tens of thousands of land mines were left buried over millions of square meters. In the decades that followed, several hundred civilians were seriously injured or killed each year while attempting to cross or farm the mined land. For many years, local volunteers and international aid workers shared the dangerous task of going meter-by-meter through the minefields, clearing them of bombs.

Along with traditional approaches such as the use of metal detectors to find buried explosives, one enterprising organization trained giant African pouched rats to detect the scent of the bombs and signal to their handlers that a mine was nearby. Because the rats weigh only about 1 to 2 kilograms, they can scamper across minefields safely, without detonating the devices. One trained rat can check an area of 200 square meters in about 30 minutes—a job that might take several days for a human using a metal detector, partly because the device would be distracted by stray pieces of scrap metal, which the rats ignore ([Kristof, 2015](#)). In the span of one year (2009), a team of 34 rats and 50 handlers cleared nearly 200,000 square meters, finding 75 land mines ([Poling et al., 2010](#)). By 2015, Mozambique officially declared that it was free of land mines. The rats are still being used in nations such as Angola and Cambodia, where other conflicts have left in their wake numerous unexploded land mines and other buried ordnance.

How do these mine-detecting rats learn their jobs? The training is intensive, and it typically takes 9 months for a rat to be “accredited” and deployed in the field, but the basic idea is simple. The rats learn that, when they detect a target scent (here, the odor of explosives) and make a specific behavioral response (such as pawing at the ground) to alert the handler, they may earn a small reward (such as a piece of banana). The rats like the treats, so they willingly scamper across fields to seek out the target smell and signal its presence in order to earn more rewards.

This kind of learning is an example of **operant conditioning**, the process whereby organisms learn to make or to refrain from making certain responses in order to obtain or avoid certain outcomes. The learning is called “operant” because the organism “operates” on the environment in a way that causes an outcome to occur. Operant conditioning is sometimes also called *instrumental conditioning*, meaning that the organism’s behavior is “instrumental” in producing the outcome.



Howard Burditt/Reuters/Newscom

Giant African pouched rats have been trained to sniff out buried land mines left over from the civil war in coastal Mozambique. Guided by little harnesses, the rats scamper around the minefield without detonating the mines and give a decisive scratch with both forepaws when they smell an explosive. The rats are trained using the principles of operant conditioning. Why do you think the animals are willing to do this work?

operant conditioning

The process whereby organisms learn to make or to refrain from making certain responses in order to obtain or avoid certain outcomes.

Operant conditioning sounds deceptively simple, but the real-world applications are complex and profound. In addition to mine detection, African pouched rats are also being trained to sniff sputum samples from patients for evidence of tuberculosis in parts of the world where locals cannot easily afford expensive laboratory testing ([Mgode, Cox, Mwimanzi, & Mulder, 2018](#)).

Closer to home, you may frequently see service dogs that help visually impaired human companions navigate busy city streets or sniffer dogs that search for drugs and explosives at airports and border crossings. One innovative group of researchers has even trained honeybees to signal when they detect the scent of illicit drugs ([Schott, Klein, & Vilcinskas, 2015](#)), raising the possibility that at some point in the future, police officers on routine traffic stops might carry along a box of bees. By the end of this chapter, you should have a fairly good idea of how all these animals learned their trades.

5.1 Behavioral Processes

Humans have been using the principles of operant conditioning as long as there have been sheep to herd, horses to ride, and puppies to housebreak. And parents throughout history have used operant conditioning when praising or rewarding their children for desired behavior and when scolding or punishing their children for misbehavior. But it was not until the end of the nineteenth century (about the same time that Ivan Pavlov was “discovering” classical conditioning) that Edward Thorndike first tried to systematically explore *how* animals learn new behaviors.

The “Discovery” of Operant Conditioning

[Chapter 1](#) describes Edward Thorndike and his studies of how cats learned to escape from puzzle boxes, such as the one shown in [Figure 5.1A](#). The puzzle boxes were made from fruit crates, each with a door that could be opened from the inside if the animal executed the correct sequence of pressing levers, pulling ropes, and stepping on pedals ([Thorndike, 1898, 1911, 1932](#)). When Thorndike would put a cat in such a box for the first time, the animal would hiss, claw at the walls, and generally try to fight its way free. Eventually, the cat would accidentally perform the movements needed to open the door and get out. Thorndike recorded how long it took the animal to escape and then returned the cat to the box to try again. After a few experiences in the box, a cat would typically learn an efficient sequence of moves that allowed it to escape. [Figure 5.1B](#) shows the data from one cat; after a dozen or so trials in the box, the cat was able to get out almost immediately.

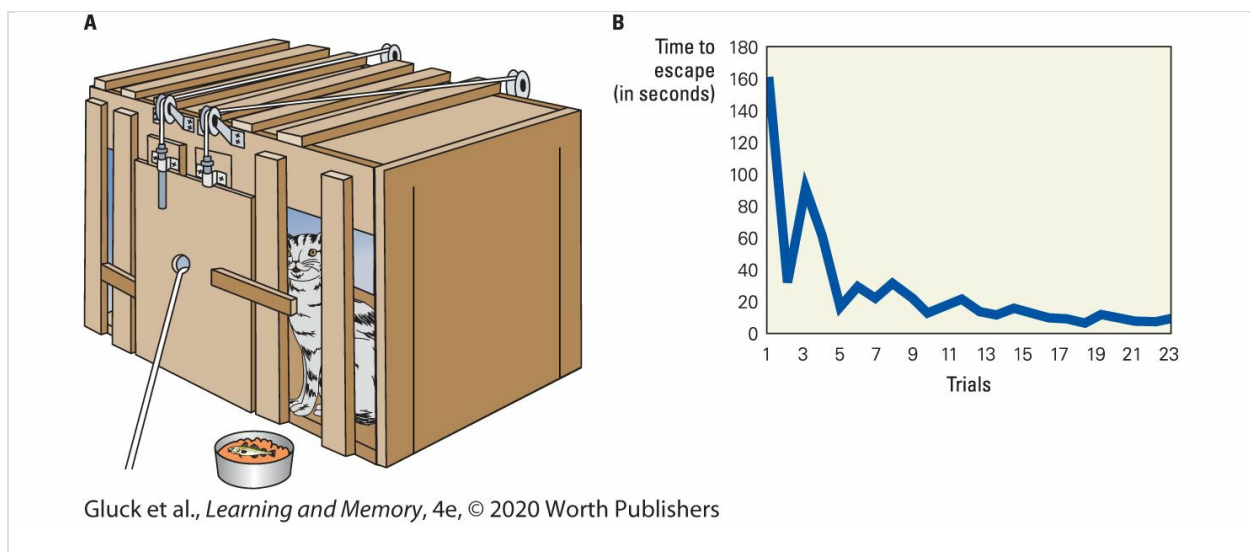


FIGURE 5.1 Thorndike's studies of animal learning (A) One of Thorndike's puzzle boxes. (B) Data from one cat that learned to escape from the puzzle box efficiently after a few experiences.

Data from [Thorndike, 1911](#).

Description

The first illustration on the left labeled A shows a cat kept inside a rectangular puzzle box. The cat is peeping out of the puzzle box and looking at a bowl of food is kept in front of the box.

The second illustration on the right shows a line graph, with peaks. The horizontal axis plots Trails and vertical axis plots Time to escape (in seconds). The data in the graph are as follows. The value of the graph starts from the point (1, 160). It suddenly decreases to a point (2, 100) and again increases to a point (5, 20). As the number of trials increased, the time taken decreases. Further, the curve rises up and down continuously and ends at point (23, 1). All data are approximate.

Thorndike concluded that when an animal's response was followed by a satisfying outcome (such as escaping from a puzzle box or obtaining food), the probability of that response occurring again in the future would increase. Thorndike formalized this idea in his *law of effect*, which is presented in [Chapter 1](#). Specifically, in the presence of a particular stimulus, called the **discriminative stimulus (S^D)**, a particular response (R) may lead to a particular outcome (O).

discriminative stimulus (S^D)

In operant conditioning, a stimulus indicating that a particular response (R) may lead to a particular outcome (O).

S^D is called a “discriminative stimulus” to emphasize that it helps the organism “discriminate” the conditions under which R will lead to O. If the outcome O is desirable or pleasant, the response R will tend to increase in frequency, strengthening the $S^D \rightarrow R$ association and making it more likely that S^D will evoke the same R in the future.

But as [Chapter 1](#) also describes, Edward Tolman argued that Thorndike's $S^D \rightarrow R$ framework was too limiting; Tolman believed that animals make responses because they (in some sense) understand that response R leads to a specific, predicted outcome O. More recent studies have supported many of Tolman's ideas and have even identified places in the brain where $S^D \rightarrow R$ associations may be stored and where expected outcomes O are processed (more on that later, in [Section 5.2](#)). For now, we will formalize operant conditioning as a three-part association:

Discriminative stimulus $S^D \rightarrow$ Response R $\rightarrow$ Outcome O

In the case of a puzzle box, S^D is the box, R is the sequence of movements needed to open the door, and O is the escape. The $S^D \rightarrow R$ association is strengthened when R is followed by a desirable outcome O.

Classical Versus Operant Conditioning

A few decades after Thorndike published his original account of operant conditioning, B. F. Skinner read it, along with Pavlov's work on classical conditioning, and concluded that these two types of learning are fundamentally different ([Skinner, 1938](#)). In classical conditioning (described in [Chapter 4](#)), organisms experience an outcome (the unconditioned stimulus, or US) whether or not they perform the conditioned response (CR). But in operant conditioning, the outcome O depends on whether the organism performs the response R.

For example, in classical conditioning of the eyeblink response, a rabbit may hear a tone (the conditioned stimulus, or CS) that is reliably followed by an airpuff US, and the rabbit may learn to make an eyeblink CR to the tone. The airpuff follows the tone whether or not the CR occurs—so this paradigm is classical conditioning. By contrast, a cat placed in a puzzle box (S^D) must learn to make a series of responses (R) in order to escape and obtain food (O). If the responses R are not performed, the outcome O does not occur. Therefore, this paradigm is operant conditioning. When deciding whether a paradigm is operant or classical, focus on the outcome. If the outcome occurs regardless of responding, the paradigm is classical; if the outcome is contingent on a response, the paradigm is operant.

Differences aside, operant and classical conditioning share many characteristics, including a negatively accelerated learning curve: [Figure 5.1B](#) shows that the time to escape from a puzzle box decreases rapidly in the first few trials and then levels off; similar leveling off occurs in classical conditioning (as seen in [Figure 4.8](#)). Both operant and classical conditioning also show extinction: a tendency for learned responses to extinguish if no longer followed by the outcome. (Extinction is discussed in more detail in [Chapter 4](#).)

TEST YOUR KNOWLEDGE

Is It Classical or Operant?

In classical conditioning, the outcome (US) follows the stimulus (CS) whether or not a learned response (CR) is performed. In operant conditioning, the outcome (O) follows the discriminative stimulus (S^D) only if a particular response (R) is performed. Analyze the following scenarios to check whether you understand the difference. ([Answers appear at the end of](#)

[the chapter.](#))

1. Since retiring, Jim spends a lot of time sitting on his back porch, watching the birds and whistling. One day, he scatters crumbs, and birds come and eat them. The next day, he sits and whistles and strews crumbs, and the birds return. After a few days, as soon as Jim sits outside and starts whistling, the birds arrive.
2. Shevonne's dog Snoopy is afraid of thunder. Snoopy has learned that lightning always precedes thunder, so whenever Snoopy sees lightning, he runs and hides under the bed.
3. Michael has accepted a new job close to home, and now he can walk to work. On the first morning, there are clouds in the sky. It starts to rain while Michael is walking to work, and he gets very wet. The next morning, there are again clouds in the sky. Michael brings his umbrella along, just in case. When it rains, he stays dry. After that, Michael carries his umbrella to work anytime the sky looks cloudy.
4. In Carlos's apartment building, whenever someone flushes the toilet, the shower water becomes scalding hot, causing him to flinch. Now, whenever he's in the shower and hears the noise of flushing, he automatically flinches, knowing he's about to feel the hot water.

Free-Operant Learning

[Chapter 1](#) introduces B. F. Skinner, the “radical behaviorist.” Skinner was attracted to Thorndike's work, with its promise of animal responses that could be measured and evaluated without requiring speculation about the animal's mental states. But Skinner thought he could refine Thorndike's techniques. Thorndike's procedures were characterized by *discrete trials*, meaning that the experimenter defined the beginning and end of each trial. For example, on one trial with the puzzle box, the experimenter would pick up a cat, put it in the box, shut the door, and record how long it took the cat to escape. He would then pick up the cat and return it to the box to start the next trial. Thorndike also built mazes (constructed out of books placed on end in various configurations) and measured how long it took animals (usually baby chicks) to find their way out. On each trial, he would place a chick at the start of the maze and record how long the chick wandered around the maze before it found its way out. He would then pick up the chick and return it to the start to begin the next trial. This type of experimental setup is often referred to as a [discrete-trials paradigm](#) because each trial is separate, or discrete, and the experimenter decides when and how often to begin a new trial.

discrete-trials paradigm

An operant conditioning paradigm in which the experimenter defines the beginning and end points of each trial.

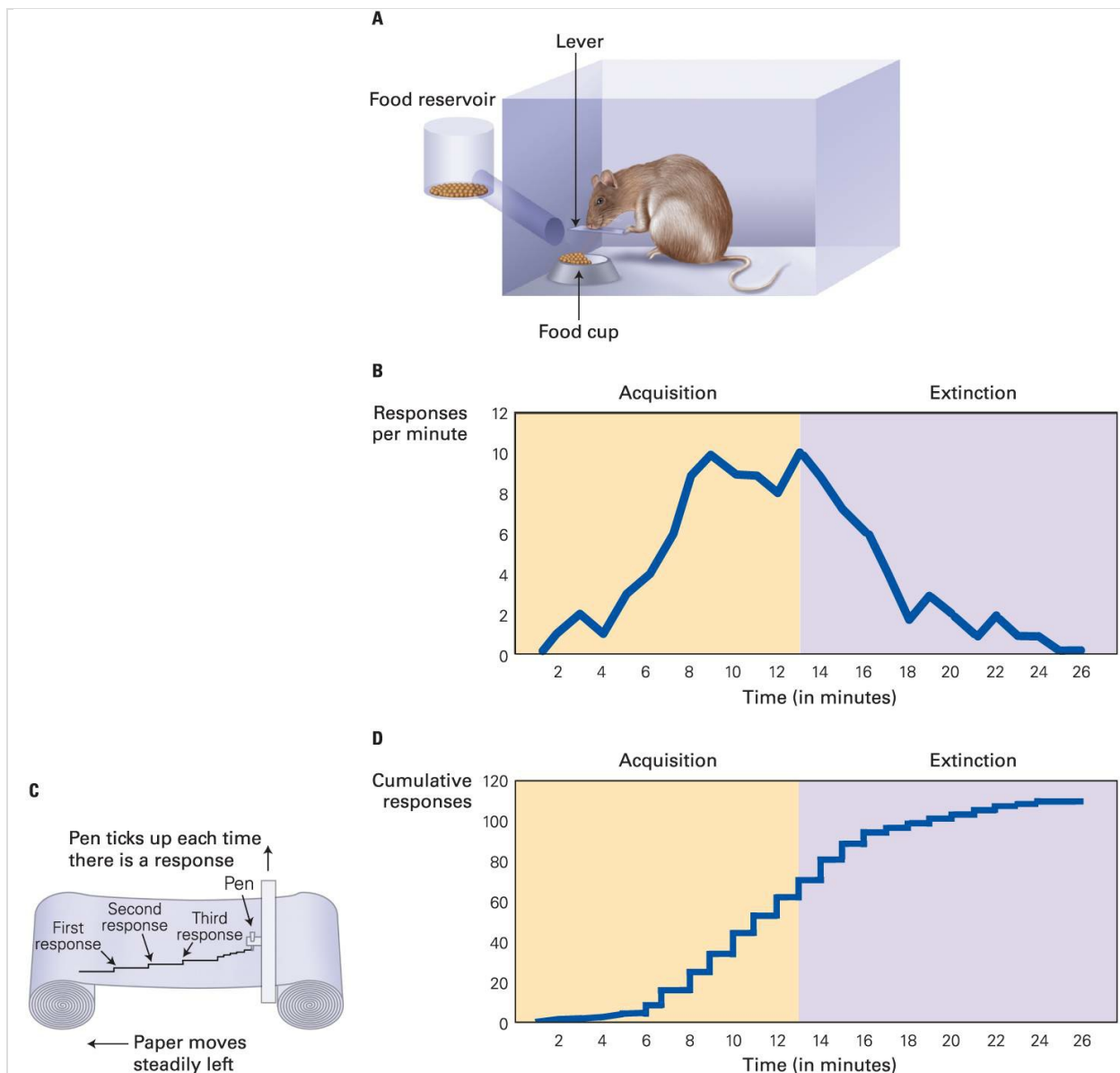
Skinner developed a maze with a return ramp so that the rat could finish one trial in the maze, collect the food, and then run back to the beginning of the maze by itself to start the next trial—and obtain the next piece of food. Part of Skinner's intention was to automate data collection so

that the experimenter no longer had to intervene at the end of each trial by returning the rat to the starting position for the next trial. But a side effect was that the animal—not the experimenter—now controlled its own rate of responding, by how quickly or slowly it ran around to start the next trial. This type of setup is often referred to as a **free-operant paradigm**, meaning that the animal can operate the apparatus freely, whenever it chooses.

free-operant paradigm

An operant conditioning paradigm in which the animal can operate the apparatus as it chooses in order to obtain reinforcement (or avoid punishment).

To measure behavior more directly, Skinner also devised a cage—now commonly called a **Skinner box**—with a trough in one wall through which food could be delivered automatically (**Figure 5.2A**). The box contained a mechanism, such as a lever or a pressure-sensitive disk, that controlled the delivery of food. When the animal pressed the lever or tapped the disk, food dropped into the trough. As the animal explored its cage, eventually it would accidentally manipulate the lever or disk and receive the food. Over time, as animals learned the relationship between the response R (pressing the lever or disk) and the outcome O (obtaining food), they would dramatically increase their rate of responding.



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

FIGURE 5.2 Operant conditioning (A) A Skinner box in which lever-press responses are reinforced by delivery of food into a food cup. (B) Hypothetical data illustrating learning by a rat in a Skinner box, shown as the mean response rate during a 26-minute experiment. During the first 13 minutes (acquisition phase), lever presses are reinforced by food delivery, so the rate of responses per minute increases; by the final (thirteenth) minute, the rat is lever-pressing almost 10 times per minute. During the last 13 minutes (extinction phase), lever presses are no longer reinforced by food delivery, so the rate of responding decreases until the rat is making 1 or fewer responses per minute. (C) A cumulative recorder provides another way to display the data from part B. Here, a paper scrolls steadily leftward, causing a pen to draw a flat line across the paper. Each time the animal makes a response, the pen moves up to a new position, jittering the line up slightly. (D) The output from a cumulative recorder, plotting the data from B. Here, the steep upward slope during the first 13 minutes (acquisition) reflects the fact that the rat is responding more and more often; the flattened slope in the final 13 minutes (extinction) shows the rate of responding is petering out. Graphs in B and D both show the same data, plotted in different ways.

Description

The illustration labeled A shows a rat in a rectangular skinner box. A rat and a bowl of food are kept inside the box. The rat is sniffing a lever in front of it. A funnel-like structure which is half-filled with food is attached to the outside the box. It is labeled Food reservoir. A bowl, labeled food cup is kept inside the box to receive the food released by the rat by pressing the lever.

A graph labeled B represents the Hypothetical data illustrating learning by a rat in the Skinner box. The horizontal axis shows Time (in minutes), and the vertical axis shows Responses per minute. The graph is divided into two planes: Acquisition and Extinction. The data in the graph are as follows. It starts at (1, 0) and slightly increases to (3, 2) on the vertical axis. Then, gradually inclines and reaches a point (9, 10). Next, it crosses the acquisition and gradually declines to a point (18, 2) in the extinction phase. Later, it steeply decreases and moves in small peaks and reaches a point from (26, 0) on the horizontal axis. All data are appropriate.

An illustration labeled C shows a cumulative recorder. It shows a spool of paper at right and left. The paper moves steadily left. A pen touching the paper ticks up each time there is a response, showing a continuously rising line made by the pen. The parts labeled, from left to right are as follows: First response, second response, third, and pen.

A line graph labeled D represents cumulative responses of a rat, in a Skinner box. The horizontal axis shows Time (in minutes), and the vertical axis shows Cumulative responses. The graph is divided into two planes: Acquisition and Extinction. The data in the graph are as follows. The values begin at point (1, 0) and gradually increase to reach (26, 110).

Skinner box

A chamber used for operant conditioning and designed so that reinforcement or punishment is delivered automatically whenever an animal makes a particular response (such as pressing or releasing a lever).

The experiment can be made a bit more elaborate by adding a discriminative stimulus S^D , such as a light on the cage wall, that signals whether response R will be reinforced; for example, pressing the lever while the light is on will trigger food delivery (outcome O), whereas lever presses while the light is off will not. Gradually, the animal learns that, in the presence of the light (S^D), lever presses (R) will result in food (O); as a result, the frequency of R when S^D is present will increase.

[Figure 5.2B](#) shows an example of the data that might be obtained from a free-operant experiment in which lever presses (R) in the presence of the light (S^D) are reinforced by food delivery (O) for the first 13 minutes of the experiment. During this acquisition period, the number of lever presses per minute increases rapidly, as the animal learns the $S^D \rightarrow R \rightarrow O$ association. This acquisition period is followed, in minutes 14 to 26 of the experiment, by an *extinction period*, meaning that the outcome (here, food delivery) no longer occurs no matter what response the animal makes. During the extinction period, the frequency of R in the presence of S^D gradually

decreases to near zero, as the animal learns the new association $S^D \rightarrow R \rightarrow \text{no O}$.

Skinner next invented a means of recording responses automatically. Back before the advent of modern computers, mechanical devices such as the one shown in [Figure 5.2C](#) recorded data on a long piece of paper rolling steadily beneath a pen. (Until fairly recently, such devices were also used for seismographs and lie detectors.) Skinner hooked up one of these devices to a Skinner box so that the pen would move up slightly to a new, higher position each time the animal responded. If the animal made no responses, the pen drew a long straight line as the paper scrolled by. But whenever the animal made a response, the pen ticked up. If the responses came quickly enough, the resulting line sloped upward steeply ([Figure 5.2D](#)). The device shown in [Figure 5.2C](#) is called a **cumulative recorder** because the height of the line at a given time shows the total number of responses that have been made in the entire experiment (cumulatively) up to that time.

cumulative recorder

A device used for recording responses in operant conditioning, designed in such a way that the height of the line it draws represents the total (cumulative) number of responses made up to a given time.

One modern example of a cumulative recorder is the odometer in a car. The odometer ticks off miles driven, and the ticks occur faster if you drive faster. When you park the car for the night and then start it up the next morning, the new mileage is added right on top of the old, for a cumulative record of total miles driven so far.

Although pen-and-paper cumulative recorders are not in general use anymore, data from operant conditioning experiments are still sometimes reported as cumulative responses (as in [Figure 5.2D](#)) instead of as response rates (as in [Figure 5.2B](#)). The label on the y-axis will tell you which kind of data you are looking at. The actual behavior being recorded is the same, whichever type of graph is used.

Learning About Stimuli and Responses

We have now defined operant conditioning as a three-part association between S^D , R, and O. But, in fact, each of these components (S^D , R, and O) can independently influence what is learned.

Discriminative Stimuli

Swimmers line up at the edge of a pool before a race. At the sound of the starting whistle, they dive in as quickly as possible to get a good start in the race. But any swimmer who dives in too early (before the starting whistle) may be penalized or even disqualified. The outcome varies, depending on whether the dive occurs before or after the whistle.

Discriminative stimuli help the learner identify (or “discriminate”) the conditions in which a response will be followed by a particular outcome. For the swimmers, the starting whistle is a discriminative stimulus signaling that dive responses will now result in a favorable outcome:

S^D (starting whistle) $\rightarrow$ R (dive) $\rightarrow$ O (good start in the race)

In a Skinner box, a light may be the discriminative stimulus. This means that lever-press responses while the light is on result in the outcome of food delivery, but lever presses while the light is off do not:

S^D (light on) $\rightarrow$ R (press lever) $\rightarrow$ O (get food)

S^D (light off) $\rightarrow$ R (press lever) $\rightarrow$ O (no food)

As the $S^D \rightarrow R \rightarrow O$ notation suggests, the discriminative stimulus is the first part of the chain that triggers the response and leads to the outcome. Sometimes, the $S^D \rightarrow R$ association is so strong that the discriminative stimulus S^D seems to evoke the learned response R automatically, no matter what other options are available. In one striking example, well-trained rats in a familiar maze ran right through a pile of food on their way to the goal box ([Stoltz & Lott, 1964](#)).

Apparently, the discriminative stimulus of the maze environment S^D was so strongly associated with the maze-running response R that unexpected food encountered along the way could not disrupt the $S^D \rightarrow R$ association. Such behavior is sometimes called a *habit slip*. People exhibit habit slips all the time, particularly when drowsy or distracted. Perhaps you have started driving to a friend’s house, only to find that your attention lapsed, and the car is now heading along the more frequently traveled route to school; or perhaps you have awoken late one morning and started hurriedly dressing for class, only to realize today is Saturday, and you can stay in bed awhile. If so, you have experienced the effects of a strong $S^D \rightarrow R$ association.

Responses

In operant conditioning, the organism learns to make a specific response R that produces a particular outcome O. A response is defined not by a particular pattern of motor actions but rather by the outcome it produces. For example, a rat in a Skinner box may receive access to food when it presses a lever:

S^D (lever in box) → R (press lever) → O (get food)

If the lever is pressed, the food arrives—whether the rat presses the lever with its left front paw, its right front paw, or even its nose. Anything the rat does to press the lever sufficiently will trigger the food delivery device. In one experiment by American psychologist Karl Lashley, rats were trained to run through shallow water in a maze, making a correct series of turns to reach the goal; later, Lashley raised the water level so that the animals had to swim. The rats continued to navigate to the goal, even though swimming involved a new set of motor responses that the rats had never executed in this environment ([Lashley, 1924](#)). Similarly, parents may reinforce a desired behavior—say, neatness—by providing an allowance to their daughter Becky if she tidies her room; the precise sequence of movements by which Becky accomplishes this task is unimportant, as long as the clothes come off the floor and the toys are put away.

But in cases this complex, how do children and animals learn which responses lead to particular outcomes in the first place? In the case of Thorndike's cats and chicks, learning occurred by trial and error; on the first trial, each animal could take minutes or hours before happening to execute the sequence of responses that opened the puzzle box door or led out of the maze. Now imagine training a service dog to help a blind man cross a busy city street. This requires that the dog approach the curb, stop, wait for a green traffic light, check for oncoming cars, and then proceed to the far curb while staying within the white painted lines. It is highly unlikely that an untrained dog would ever happen to make that complete sequence of responses and accidentally discover that the trainer would reinforce exactly those behaviors.

As you may have guessed, researchers and animal trainers who want to train complex behaviors rarely rely on accidents. Instead, they have developed special operant conditioning approaches, two of which are described next.

Shaping

A training process called [shaping](#) works by reinforcing successive approximations to the desired response. Consider the following example. When a rat is first placed in a Skinner box, it may perform any of its natural behaviors: grooming, exploring, or just sitting quietly. When the rat happens to wander near the food tray, the experimenter drops in a piece of food. The rat eats the food and starts to learn an association between the tray and food. After a few such trials, the rat starts spending time near the food tray. The experimenter then changes the rules so that, now, the rat must also be near the lever before food is dropped. Soon, the rat learns to loiter in the vicinity of the lever. Once the rat has learned this, the rules change again: food is dropped only if the animal is actually touching the lever, then only if the animal is rearing up and touching the lever, then only if the animal is pressing down on the lever. Gradually, by a series of successive approximations, the desired response is learned: the rat presses the lever to obtain food.

shaping

An operant conditioning technique in which successive approximations to the desired response are reinforced.

If you think this sounds like a difficult and time-consuming process, you're right. Shaping requires considerable skill on the part of the experimenter, who must decide how quickly to proceed, how difficult to make each new stage, and even whether to back up a few steps if the animal appears to be getting confused. Some researchers have proposed standardized methods and criteria that can help optimize shaping techniques (e.g., [Galbicka, 1994](#)). Still, the intricacies of shaping are one reason that people often pay professional animal trainers to housebreak puppies.

The principles of shaping can be useful in many situations where difficult learning objectives can be reached by successive approximation. For instance, a physical therapist can use shaping to guide a patient who has suffered a stroke and is attempting to relearn motor control. If the patient has lost the ability to use his left hand, he might first be instructed to practice simply opening and closing his hands before progressively working up to more complex tasks that require fine motor control, such as grasping and using a spoon or holding and writing with a pencil. Shaping has been used to help teach children with autism how to speak, by first reinforcing any vocalizations, then reinforcing only vocalizations that sound like words, and eventually reinforcing actual word production ([Lovaas, 1987](#)). Shaping is also used to train service animals, such as guide dogs and sniffer dogs (see [“Learning and Memory in Everyday Life,”](#) for more on how sniffer dogs are

trained and evaluated).

LEARNING AND MEMORY IN EVERYDAY LIFE

Drug-Detecting Dogs

If you've traveled by air in the past few years, then your luggage has probably been inspected by specially trained sniffer dogs, now commonly used (often behind the scenes) at airports and border crossings to help authorities detect smuggled drugs, explosives, and other illegal substances. The sniffer dogs can search baggage in a fraction of the time it would take a human. And the dogs are not only fast but sensitive; they can detect faint odors even if the target substance is sealed inside plastic or smeared with chocolate to disguise the scent.

Sniffer dogs are taught their jobs through operant conditioning. By now, you should be able to guess many of the details. Training usually starts with positive reinforcement, in which a puppy learns to fetch a toy to obtain a reward such as playtime with the trainer (a reward most dogs find highly reinforcing). Then the toy is doused with a target scent—say, the scent of heroin. Each time the dog retrieves the scented toy and gets a rewarding bout of playtime, the association between odor, retrieval, and reward is strengthened.

The trainers continue by introducing other objects doused with the same odor, so that the dog learns to retrieve any object with the target scent. Sometimes a clicker or other secondary reinforcer (defined on [page 185](#)) is introduced, so that the training session need not be interrupted for playtime after every successful retrieval. Later on, the dogs may be trained to give an “alert” response instead of actually retrieving the object, and the target scent can be expanded to include multiple odors, so that a dog will give the alert response upon sensing cocaine and other illicit drugs in addition to heroin.

How accurate are the dogs? One study of more than 1,200 tests performed in police canine training sessions reported that, on average, the dogs correctly indicated the presence of drugs in more than 90% of trials that involved detecting marijuana but only about 70% of trials with heroin ([Jezierski et al., 2014](#)). The dogs also produced false alarms in about 12% of heroin trials, giving the alert response when no drugs were present.

The false alarm rate may be even higher in “real-world” searches. A widely publicized report by the *Chicago Tribune* claimed that when police used drug-sniffing dogs to check cars pulled over during traffic stops, the dogs generated more false alarms than correct responses ([Hinkel & Mahr, 2011](#)). Such false alarms can have serious economic consequences, as when an airport is shut down for several hours after a bomb-detecting dog identifies a suspicious object that is later determined to be harmless.

Why so many false alarms? Part of the reason may simply be that drug odors can persist for up to 48 hours ([Jezierski et al., 2014](#)), so the dogs may be responding to lingering scents from drugs that are no longer present. But another possibility is that dogs respond to subtle (possibly unconscious) cues from their human handlers. In one laboratory study, where the handlers had been led to believe that the target odor was hidden in a location marked by a piece of red paper, the dogs gave significantly more alert responses in that location, even though no odor was present ([Lit, Schweitzer, & Oberbauer, 2011](#)). Furthermore, when a dog gives an alert signal in the field, the attention this signal elicits from the handler may unintentionally reinforce the signaling behavior, even if the alert later turns out to have been a false alarm.

Interestingly, similar controlled studies with giant African pouched rats (such as the mine detectors you read about in the [chapter opener](#)) suggest that the rats are much less prone to false alarms. A possible explanation is that, while dogs are highly motivated to interact with and obtain attention from their human handlers, the pouched rats seem less interested in earning praise from their handlers and more focused on how to earn the next banana reward.

Chaining

A related technique is [chaining](#), in which organisms are gradually trained to execute complicated sequences of discrete responses. Skinner once trained a rat to pull a string that released a marble, then pick up the marble with its forepaws, carry it over to a tube, and drop it inside the tube ([Skinner, 1938](#)). Skinner could not have taught such a complex sequence of responses all at once. Instead, he added “links” to the chain of learned responses one at a time: he first trained the rat to pull the string, then trained it to pull the string and pick up the marble, then trained it to pull the string and pick up the marble and carry it to the tube, and so on. Sometimes, it is more effective to train the steps in reverse order, in a process called *backward chaining*: first train the rat to drop the marble in the tube, then train the rat to carry the marble to the tube and drop it in, and so on. Either way, at each stage, the rat must perform a progressively longer sequence of responses to gain its food.



Jon-Michael Sullivan/ZUMA Press/Newscom

Twiggie is a squirrel who waterskis behind a miniature remote-controlled speedboat at a water park in Florida. How do you

think she was trained to do this trick?

chaining

An operant conditioning technique in which organisms are gradually trained to execute complicated sequences of discrete responses.

Chaining is a useful technique for training humans, too. Workers learning a manufacturing process are often taught it one step at a time ([Walls, Zane, & Ellis, 1981](#)), and trainee pilots may master landing sequences by practicing progressively longer sequences on a flight simulator ([Wightman & Sistrunk, 1987](#)).

Learning About Outcomes

So far we have discussed outcomes fairly loosely. Formally, a **reinforcer** is a consequence of behavior that leads to increased likelihood of that behavior in the future. For example, food is a reinforcer to a hungry animal, and the animal may learn to repeat behaviors that result in access to food. On the other hand, a **punisher** is a consequence of behavior that—you guessed it—leads to decreased likelihood of that behavior in the future. For instance, a rat will learn to stop pressing a lever if it receives a mild electric shock for doing so. Some children who have not stopped thumb sucking on their own agree to have their thumbs coated with a harmless but bitter-tasting substance; each thumb-sucking response then results in a nasty taste, and this punishment may decrease the rate of thumb sucking in the future.

reinforcer

A consequence of behavior that leads to increased likelihood of that behavior in the future.

punisher

A consequence of behavior that leads to decreased likelihood of that behavior in the future.

Put another way, **reinforcement** is the process of providing outcomes (reinforcers) that lead to *increased* probability of a behavior, and **punishment** is the process of providing outcomes (punishers) that lead to *decreased* probability of a behavior. Sounds simple enough, right? But the reality is more complicated, for both reinforcers and punishers.

reinforcement

The process of providing outcomes (reinforcers) that lead to increased probability of a particular behavior occurring in the future.

punishment

The process of providing outcomes (punishers) that lead to decreased probability of a particular behavior occurring in the future.

Primary Reinforcers

Food, water, sleep, comfortable temperatures, and sex are all examples of [primary reinforcers](#): they are of biological value to organisms, and therefore organisms will tend to repeat behaviors that provide access to these things or conditions. Psychologist Clark Hull's [drive reduction theory](#) proposed that all learning reflects the innate, biological need to obtain primary reinforcers ([Hull, 1943, 1952](#)). The motivation to obtain primary reinforcers was a key variable in Hull's equations—with which, as discussed in [Chapter 1](#), he hoped to explain all learning.

primary reinforcer

A reinforcer, such as food, water, or sleep, that is of biological value to an organism.

drive reduction theory

The theory, proposed by Clark Hull, that all learning reflects the innate, biological need to obtain primary reinforcers.

One complication to this theory is that primary reinforcers are not always equally reinforcing. Thirsty animals will work to obtain access to water, a primary reinforcer; but if they are not thirsty, access to water is not particularly reinforcing. In addition, not all primary reinforcers are created equal. Hungry animals will work to obtain food, but they will work even harder for food they like. For example, rats will run a maze faster for bread and milk (which they find especially tasty) than for sunflower seeds, even though the seeds satiate hunger just as effectively ([Simmons, 1924](#)).

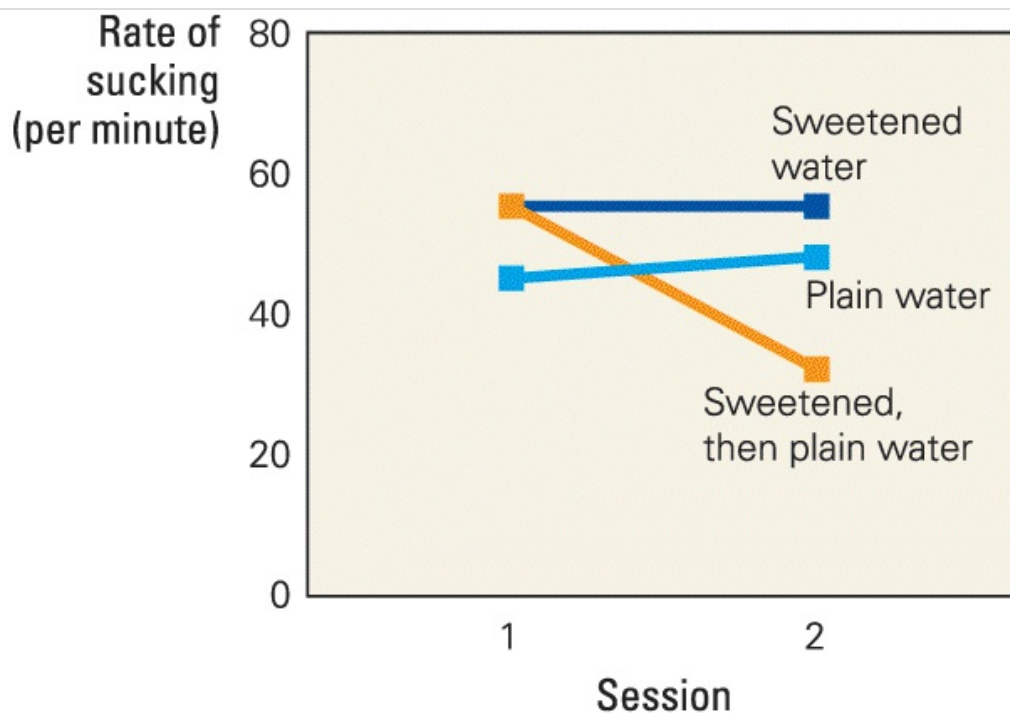
In short, the identity of the reinforcer matters. In operant conditioning, organisms learn that the response R results not just in any random outcome but in a specific outcome O. A switch in the outcome may produce a change in responding. For instance, rats can be trained to make lever-press responses to obtain either food pellets or sugared water, but they tend to prefer the sugared water. If rats are first trained to respond in order to obtain sugared water, and then the reinforcer is switched to food pellets, the rats' rate of responding plummets ([Weatherly, Plumm, Smith, & Roberts, 2002](#)). This is an example of [negative contrast](#), in which the reinforcing value of one reward is reduced because a better reward is expected ([Flaherty, 1982](#)).

negative contrast

The phenomenon in which the reinforcing value of one reward is reduced because a better reward is expected.

Negative contrast can be observed in humans, too. In one classic study, infants were given access to an artificial nipple that they could suck to obtain water. Some infants received plain water in sessions 1 and 2 ([Figure 5.3](#), light blue line), while others received sugared water in both

sessions (dark blue line). Unsurprisingly, the infants who were given sweetened water sucked more vigorously: plain water is an acceptable reinforcer, but sweetened water is a preferred reinforcer. But a third group of infants was given sweetened water in session 1 and then switched to plain water in session 2. As shown in [Figure 5.3](#) (orange line), these infants' responses plummeted after the switch—to a lower level than those infants who had received plain water all along ([Kobre & Lipsitt, 1972](#)). Similar negative contrast effects can be observed in children who go trick-or-treating expecting candy but instead get raisins (which on normal days they might enjoy), as well as in game-show contestants who hope to win the million-dollar grand prize but instead dejectedly settle for the consolation prize of a free vacation, which they would normally be delighted to accept.



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FIGURE 5.3 The negative contrast effect A normally acceptable reinforcer evokes less responding if a preferred reinforcer is expected: human infants will suck at a higher rate for sucrose-sweetened water (dark blue) than for plain water (light blue), indicating that sweetened water is a preferred reinforcer. But a third group of infants, who received sweetened water in session 1 and were then switched to plain water in session 2 (orange line), sucked less vigorously in session 2 than the infants who received plain water all along.

Data from [Kobre & Lipsitt, 1972](#).

Description

The horizontal axis shows Session, and the vertical axis shows the Rate of sucking (per minute). The data in the graph are as follows.

Three graph lines are shown, as follows.

Sweetened water: 60 sucks per minute in Session 1 and 60 sucks per minute in Session 2. Plain water: 44 sucks per minute in Session 1 and 46 sucks per minute in Session 2. Sweetened, then plain water: 60 sucks per minute in Session 1 and 32 sucks per minute in Session 2.

Secondary Reinforcers

In addition to primary reinforcers such as food and water, learning can also be driven by [secondary reinforcers](#), which are reinforcers that initially had no biological value but have been paired with (or predict the arrival of) primary reinforcers ([Shahan, 2010](#)). The best example of a secondary reinforcer is money. Money itself has no biologically reinforcing properties, but it can be exchanged for any number of primary reinforcers, including food, shelter, and even sex (millionaires tend to attract potential mates much more easily than paupers do). For a student, grades may function as secondary reinforcers, if they are paired with parental approval and other pleasant outcomes.

secondary reinforcer

A reinforcer that initially has no biological value but that has been paired with (or predicts the arrival of) a primary reinforcer.

Secondary reinforcement is often used in prisons, psychiatric hospitals, and other institutions to motivate inmates or patients to respect rules or perform chores such as making beds. Each desired behavior is reinforced with a token, and tokens can then be exchanged for privileges (say, access to the telephone or group activities). Such arrangements are called [token economies](#) because the tokens function the same way as money does in the outside world ([Hackenberg, 2009](#)). Token economies have been used with some success to modify behavior in children with intellectual disability or with autism ([Matson & Boisjoli, 2009](#)); even nonverbal children may be able to learn that certain responses result in acquisition of tokens, which in turn can be exchanged for candy or toys.

token economy

An environment (such as a prison or schoolroom) in which tokens function the same way as money does in the outside world.

Animals, too, will work for secondary reinforcers. For example, trainers can use secondary reinforcement to teach dolphins to do tricks. The trainer first pairs a whistle sound with food reinforcement until the dolphin has learned an association between the whistle and food; at this point, the whistle has become a secondary reinforcer, and it can be used to maintain the behavior ([Pryor, Haag, & O'Reilly, 1969](#)). Horse trainers often use a similar technique, pairing a clicking noise with oats and then eventually using the clicking noise alone ([Skinner, 1951](#)). In both cases, as long as the secondary reinforcer is occasionally followed by food, the behavior is maintained. Secondary reinforcers are particularly useful in animal training because the animal trainer can deliver clicker reinforcement immediately and from a distance; the more quickly the response is followed by reinforcement, the more effective the reinforcement is.

A standard explanation of secondary reinforcement is that, by virtue of being paired with a primary reinforcer (such as food), secondary reinforcers (such as the clicker) become reinforcers themselves, which the organism will work to obtain. Some evidence supports this view. However, other research suggests that animals are not “fooled” into thinking that the secondary reinforcers are worth working to obtain; rather, the secondary reinforcers provide informational feedback that behavior is on the right track for obtaining the primary reinforcer: “Keep executing this response, and you’ll eventually get food” ([Shahan, 2010](#)). This is similar to a prison inmate who completes chores not because she is particularly motivated to obtain the tokens themselves but because she knows that acquiring tokens is a way to obtain things that she does like.

Remember the giant African pouched rats, mentioned in the introduction to this chapter, that have been trained to sniff out buried mines? They are trained through a combination of secondary reinforcement and shaping ([Poling et al., 2010, 2011](#)). Young rats are trained (through classical conditioning) to associate the popping sound of a clicker with a small reward (such as banana pulp). The food is a primary reinforcer, and the clicker sound becomes a secondary reinforcer that predicts the food. Next, the rats are placed in a cage that contains a hole in the floor, below which the scent of an explosive (such as TNT) is placed. The rats are trained to hold their noses in the hole for a few seconds in order to earn clicker reinforcement. Then the rats are trained with multiple holes, only some of which are TNT-scented, following which the rats are reinforced for correctly performing the “sniff and hold” response to the scented holes but not to the unscented ones:

S^D (odor of TNT) → R (sniff and hold) → O (clicker)

S^D (no odor of TNT) → R (sniff and hold) → O (no clicker)

The clicker is particularly useful here because the trainers can provide the reinforcement immediately, without waiting for the rat to finish responding and retrieve a treat. The clicker sound itself is still occasionally paired with food so that it remains reinforcing.

Eventually, the rats are moved outside, where they can run around small yards that contain buried TNT-scented targets, and finally they are trained to work methodically up and down marked paths in an actual simulated minefield. Only after a rat can successfully detect every simulated mine in a 200-square-meter test area containing 5 to 7 targets is the animal “licensed” as an operational mine-detection animal.

The same general techniques are used to train giant pouched rats to detect tuberculosis in human sputum samples, as described at the start of this chapter. The key here is that infected samples emit an odor that the rats’ highly sensitive olfactory system can recognize. The odor of infection becomes the discriminative stimulus S^D, triggering the “sniff and hold” response R, and resulting in clicker reinforcement O (which is sometimes paired with food):

S^D (odor of infected sample) → R (sniff and hold) → O (clicker)

After several weeks of training, rats can move rapidly down a row of sample wells, pausing for clicker reinforcement when they sniff out an infected sample—thus signaling to the human observer which of the samples likely came from infected patients. Whereas human microscope technicians can process only a few dozen samples per day, highly trained rats can process (sniff at) 140 samples in about 20 minutes, and studies suggest that the rats’ diagnoses are more than 90% correct ([Weetjens et al., 2009](#)). As mentioned at the beginning of the chapter, trained rats are currently being used in clinics in Tanzania and Mozambique, and some studies suggest they can even identify infected samples that are “missed” by standard lab tests alone ([Mgode et al., 2018](#)).

Punishers

Just as organisms will work to obtain reinforcers, they will also work to avoid punishers. Common punishers for animals include pain, confinement, and exposure to predators (or even the scent of predators). Common punishers for humans are monetary fines, social disapproval,

and jail time.

As discussed in [Chapter 1](#), [Thorndike \(1911\)](#) originally assumed that punishers were simply the inverse of reinforcers: whereas reinforcement increases the probability that a response will occur again in the future, punishment decreases that probability. Later, both [Thorndike \(1932\)](#) and [Skinner \(1938, 1953\)](#) concluded that punishment is not nearly as effective as reinforcement at controlling behavior. In the end, Thorndike dropped the idea of punishment from his law of effect, concluding that while reinforcement does indeed increase the probability that a response will be repeated in the future, the effects of punishment are erratic and unreliable and can even at times result in paradoxical *increases* in the punished behavior ([Postman, 1962](#); [Thorndike, 1943](#)).

Nevertheless, many modern researchers argue that punishment can indeed be very effective in modifying behavior (for review, see [Staddon, 1995](#)). The problem is that several factors determine how effective the punishment will be. We describe four of the most important factors here.

1. Punishment leads to more variable behavior.

According to the law of effect, reinforcement of a particular response R increases the probability that the same response R will occur in the future. In contrast, punishment of R decreases the probability that R will occur in the future but does not predict what response will occur instead of R! In fact, punishment tends to produce variation in behavior, as the organism explores other possible responses. That is fine if the primary goal is simply to eliminate an undesired response (such as training a child not to go near a hot stove). But it is not a particularly good way to train desired behaviors. If the goal of conditioning is to shape behavior in a predetermined way, then reinforcing the desired response generally produces much faster learning than simply punishing alternative, undesired responses.

2. Discriminative stimuli for punishment can encourage cheating.

Discriminative stimuli can signal whether an operant response will be reinforced, and they can also signal whether a response will be punished. This means that the absence of the

discriminative stimulus might signal that the response will *not* be punished. For example, to a speeding driver, the sight of a police car is a discriminative stimulus for punishment: speeding in the presence of this stimulus will probably be punished. But speeding in the absence of a police car will probably not be punished. In this case, punishment does not train the driver to drive slowly; it only teaches him to suppress speeding in the presence of police cars. When no police car is visible, speeding may resume. Similarly, the dominant male in a group of chimpanzees may punish females for mating with any other males—but when his back is turned, females often sneak off into the bushes with lower-ranking males. And rats that have been painstakingly trained to eat no more than four pellets at a time will often eat all the food in sight if no human is watching ([Davis, 1989](#)).

3. Concurrent reinforcement can undermine the punishment.

The effects of punishment can be counteracted if reinforcement occurs along with the punishment. Suppose a rat first learns to press a lever for food but later learns that lever presses are punished by shock. Unless there is another way to obtain food, the rat is likely to keep pressing the lever to obtain food reinforcement, in spite of the punishing effects of shock. Similarly, a child may be reprimanded for talking in class, but this punishment will be much less effective if the behavior is simultaneously reinforced by approval from classmates. And although a speeding driver risks a hefty ticket, the effects of this punisher may be counteracted by the reinforcing fun of driving fast.

4. Initial intensity matters.

Punishment is most effective if a strong punisher is used from the outset. In one study, rats received a shock as they ran through a maze to the goal box ([Brown, 1969](#)). The shock was initially delivered at the lowest intensities (1 or 2 volts) and had little effect on behavior. Gradually, across several days, shock intensity increased to 40 volts. Behavior was essentially unaffected in these rats, even though naive rats given a 40-volt shock would stop running immediately. Apparently, early weak shocks made the rats insensitive to later, stronger shocks. The effectiveness of the strong shock was completely undermined by starting weak and working up from there. Similarly, a child who misbehaves in class may first receive a warning, then a scolding, then detention, then expulsion. The expulsion, when it comes, may be much less

effective at deterring future misbehavior because of the prior milder punishments. Likewise, a speeding driver may not be deterred much by a hefty \$500 ticket if she is already accustomed to paying lesser fines. In each case, the prior weak punishers may undermine the effectiveness of the severe punisher, when it finally comes. (See [“Learning and Memory in Everyday Life”](#) for more discussion of the problems with punishment.)

LEARNING AND MEMORY IN EVERYDAY LIFE

The Problem with Punishment

Around the world, about 80% of children are occasionally spanked or otherwise physically punished by their parents as a way to discourage unwanted behaviors ([UNICEF, 2014](#)). Spanking is a form of punishment that even a very young child can understand; it is easy to administer, and it can effectively reduce the frequency of an unwanted response.

But physical punishment is controversial. Many people believe that hitting a child is never justifiable. Some studies have suggested that children who are spanked can develop emotional problems, including aggression and stress ([Gershoff & Grogan-Kaylor, 2016](#); [Gershoff, Sattler, & Anasari, 2017](#)), whereas other studies have found that occasional mild spanking does not cause any lasting harm ([Ferguson, 2013](#)).

Parents who want to avoid spanking have other options. Punishment does not have to cause physical pain to be effective. Scolding is a form of punishment that does not cause physical harm; other methods are time-out, grounding, and withholding of allowance.

But there is still the problem that punishment is hard to apply effectively. Take a hypothetical example. Shawn has two busy working parents and several older siblings. When Shawn is well behaved, his siblings tend to get most of the parental attention; but when Shawn breaks china, fights with his brothers, or causes trouble at school, the parental spotlight shines on him. Although they may think they are punishing Shawn, his parents are actually reinforcing his bad behavior by giving him attention when he misbehaves.

What can be done? For one thing, Shawn’s parents should punish unwanted behavior with a minimum of fuss so that the offender gets less attention—and less reinforcement—for it. They can minimize opportunities for the unwanted behavior (such as by keeping the china on a higher shelf). They can also reduce unwanted behavior by differential reinforcement of alternative behaviors, praising Shawn on the days when he does not misbehave. This means that Shawn’s parents have to commit the extra time and effort to pay more attention to their youngest child, not by merely punishing him when he is bad but by also acknowledging him when he is good. The payoff may be a well-behaved child and a happier family, while avoiding many of the problems of punishment.

Differential Reinforcement of Alternative Behaviors

Given the problems with punishment, many prefer using the carrot rather than the stick. Instead of delivering punishment each time an unwanted behavior is exhibited, one can reward preferred alternative behaviors—a process known as [differential reinforcement of alternative behaviors](#)

(abbreviated [DRA](#)). For example, some children with autism or developmental disorders show persistent habits of self-injurious behavior, such as repeatedly banging their head against the wall or biting their own hand. Rather than punishing the child for each instance of the unwanted behavior, parents or therapists can reward instances of desired behavior, such as compliance with instructions to complete a task or eat a nourishing meal ([Petscher, Rey, & Bailey, 2009](#)). DRA can work particularly well if the rewarded behavior is incompatible with the unwanted behavior. For instance, as long as she is sitting in a chair to complete homework or eat a meal, the child is physically unable to bang her head against the wall.

differential reinforcement of alternative behaviors (DRA)

A method used to decrease the frequency of unwanted behaviors by instead reinforcing preferred alternative behaviors.

In 2011, the movie theater chain Cinemark introduced a mobile phone application that allows customers to check movie listings and buy tickets. It also tried a new tactic to combat a pernicious feature of the modern cinema experience: fellow moviegoers who text during the performance, creating phone screen light that may distract those seated nearby. Warnings at the start of the film to put away cell phones, threats to eject the disobedient, and even social disapproval all having failed, Cinemark turned to reinforcement of an alternate behavior: users who activate the phone app at the start of the movie and don't use the phone again for the rest of the movie earn coupons—secondary reinforcers that can be exchanged for rewards like free popcorn and soda. The jury is still out on whether this attempt at DRA actually reduced texting-while-viewing, compared with more traditional punishment-based approaches.



Wavebreakmedia/Getty Images

What types of social cues can serve as punishers for unwanted public behavior?

Putting It All Together: Building the $S^D \rightarrow R \rightarrow O$ Association

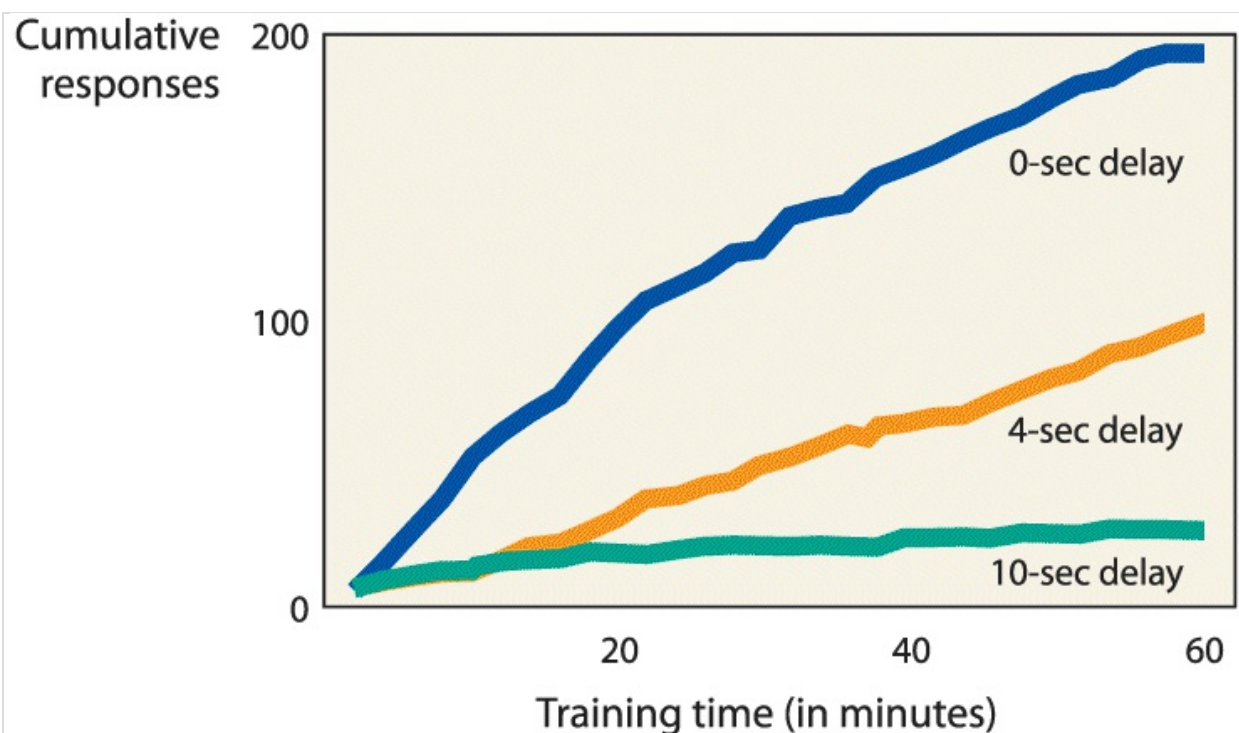
The components of operant learning—the discriminative stimulus, response, and outcome—can be arranged in different ways. An experimenter can vary factors such as the temporal spacing between R and O, whether the outcome is something added to or subtracted from the environment, and even the regularity with which the outcome follows the response.

Timing Affects Learning

In most of the operant conditioning examples presented so far, the outcome (reinforcer or punisher) has immediately followed the response. For instance, as soon as the rat presses a lever, food drops into the Skinner box; as soon as a pouched rat locates a buried land mine, the trainer

provides clicker reinforcement; as soon as toddler Annie starts to suck her thumb, the bitter taste on her finger reminds her to stop.

Normally, immediate outcomes produce the fastest learning. If there is no delay between response and reinforcement, then the odds are good that the most recent behavior will be identified as the response that caused the outcome, and the frequency of that response will increase. But if there is a long delay, it is more likely that other behaviors have taken place during the interval, and those behaviors (instead of the earlier response) might thus be associated with the outcome. To illustrate this idea, [Figure 5.4](#) shows that rats learn a lever-pressing task quickly when the delay between response and food delivery is 0 seconds, but they are slower to learn the association if the delay is 4 seconds, and even slower if the delay is lengthened to 10 seconds ([Schlinger & Blakely, 1994](#)).



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FIGURE 5.4 Effects of a delay between a response and an outcome Rats were trained to lever-press, with food reinforcement delivered immediately or after a short delay. The rats learned more quickly with no delay than with a 4-second delay. They learned more slowly still with a 10-second delay, as indicated by the nearly flat cumulative response curve.

Information from [Schlinger & Blakely, 1994](#).

Description

The horizontal axis shows Training time (in minutes). The vertical axis shows Cumulative responses. The data in the graph are as follows.

0-second delay. The line starts at 0 and increases steeply to 200.

4-second delay. The line starts at 0 and increases to 100

1-second delay. The line starts at 0 and slightly increases to 10.

Temporal contiguity of response and outcome has a similar impact on the effectiveness of punishment. Unfortunately, human society often employs delayed punishment. Criminals may not come to trial—much less serve their sentence—until months or years after committing the crime. A middle school student who misbehaves in the morning and receives detention after school experiences a delay of several hours between response and outcome. Such delays undermine the punishment’s effectiveness and may weaken learning.

Outcomes Can Be Added or Subtracted

Suppose Becky learns that, at dinnertime (S^D), helping to set the table (R) will result in the reinforcement of parental praise (O). Her younger sister Annie learns that, after a bitter coating has been applied to her thumb (S^D), sucking her thumb (R) will trigger an unpleasant taste (O). Note that in both these examples, the outcome is something “added” to the child’s environment:

S^D (dinnertime) $\rightarrow$ R (set the table) $\rightarrow$ O (praise)

S^D (tastant on thumb) $\rightarrow$ R (thumb sucking) $\rightarrow$ O (bitter taste)

These paradigms are technically called **positive reinforcement** and **positive punishment**. Note that here the word *positive* does not mean “good”; instead, it means “added” in the mathematical sense (as in a positive number). In positive reinforcement, the desired response causes the *reinforcer* to be added to the environment; in positive punishment, an undesired response causes a *punisher* to be added to the environment.

positive reinforcement

A type of operant conditioning in which the response causes a reinforcer to be “added” to the environment; over time, the

response becomes more frequent.

positive punishment

A type of operant conditioning in which the response causes an undesirable element to be “added” to the environment; over time, the response becomes less frequent.

There are also learning situations in which the outcome is the *removal* of something from the environment. In **negative reinforcement**, behavior is encouraged (reinforced) because it causes an undesirable element to be removed from the environment. Here, the word *negative* means “subtracted” in the mathematical sense (as in a negative number). For example, if you have a headache, you can take aspirin to make the headache go away:

S^D (headache) → R (take aspirin) → O (no more headache)

negative reinforcement

A type of operant conditioning in which the response causes an undesirable element to be “subtracted from” the environment; over time, the response becomes more frequent.

The net result is that you are more likely to take aspirin again the next time you have a headache, so this scenario is an example of reinforcement. The outcome is not something added to your environment but something subtracted—that is, the headache is taken away—so this is negative reinforcement.

Similarly, a rat can be placed in a chamber with an electrified floor grid that administers electric shocks. The rat can escape these shocks by climbing onto a wooden platform. In this case, the response is climbing, and the outcome is escaping from the shock:

S^D (shock) → R (climb) → O (no more shock)

The net result is that the rat is more likely to climb the platform in the future. In other words, the climbing response has been reinforced. Because the outcome involves a subtraction (i.e., shock is subtracted, or taken away), this is an example of negative reinforcement. Negative reinforcement is sometimes called *escape* or *avoidance* training because the response causes an escape from, or avoidance of, something aversive (such as a headache or shock).

Just as behavior can be reinforced by taking away bad things, so can behavior be punished by taking away good things. This kind of paradigm is called **negative punishment** because something is *subtracted* (taken away) from the environment, and this subtraction punishes the

behavior. (Negative punishment is also sometimes called *omission training* because the response R results in something being “omitted from” the environment.) For example, if Becky displays aggressive behavior toward other children during recess, the teacher may make Becky sit by herself while the other children play:

S^D (recess) → R (aggressive behavior) → O (loss of playtime)

negative punishment

A type of operant conditioning in which the response causes a desirable element to be “subtracted from” the environment; over time, the response becomes less frequent.

The net effect is that Becky may be less likely to display aggressive behavior in the future. This kind of negative punishment is sometimes called a *time-out*: Becky is punished by time away from a normally reinforcing activity. Time-outs work only if the activity being restricted is something reinforcing. A time-out from an activity the child dislikes may actually serve to reinforce, rather than reduce, the bad behavior that earned the time-out!

Negative punishment is widely applied in human society: teenagers may be grounded for staying out too late, drivers may have their licenses suspended for speeding, and customers who do not pay their phone bills on time may have their service discontinued. In each case, an undesirable behavior is punished by revoking privileges, in the hope of decreasing the likelihood that such behavior will occur again in the future.

Table 5.1 summarizes the four types of training. Keep in mind that the terms *reinforcement* and *punishment* describe whether the response increases in frequency (reinforcement) or decreases in frequency (punishment) as a result of training. The terms *positive* and *negative* describe whether the outcome is something added (positive) or something taken away (negative).

TABLE 5.1 Operant conditioning paradigms

	Response increases (reinforcement)	Response decreases (punishment)
Outcome is added (positive)	Positive reinforcement Example: Clean room → get weekly allowance	Positive punishment Example: Exceed the speed limit → ticket and fine
Outcome is removed (negative)	Negative reinforcement (escape/avoidance training)	Negative punishment (omission training)

Example: Take aspirin → headache goes away

Example: Fight with other children → time-out from play

Laboratory experiments often fit neatly into the grid shown in [Table 5.1](#), but real life is more complicated. Sometimes it is difficult to determine whether an individual is learning based on reinforcement or punishment or both. For example, when studying for an exam, are students working to obtain a good grade (positive reinforcement) or to avoid flunking (negative reinforcement)? It could be either—or both. Similarly, a child who misbehaves may receive both a scolding (positive punishment) and a time-out (negative punishment), and both considerations may motivate her to keep bad behavior to a minimum.

TEST YOUR KNOWLEDGE

Reinforcement Versus Punishment

It is easy to confuse the ideas of negative reinforcement, positive punishment, and so on because in other contexts we often use the words *positive* and *negative* to mean “good” and “bad.” Don’t fall into this trap! First ask yourself whether the outcome is added to (positive) or subtracted from (negative) the environment, and then ask yourself whether the outcome causes a behavior to increase (reinforcement) or decrease (punishment).

Try your hand at each of the following scenarios and see if you can tell whether it is an example of positive reinforcement, negative reinforcement, positive punishment, or negative punishment. In each case, answer these questions in order to decide: (a) Who does the learning? (b) What is the response (the behavior that is being altered)? (c) What is the outcome? (d) What is the discriminative stimulus that determines whether the response will produce that outcome? (e) Is the outcome something added or taken away? (f) Does the response increase or decrease as a result of learning? ([Answers appear at the end of the chapter.](#))

1. At the grocery store, 2-year-old Lucy sees candy and wants it. Her mother Susan says no, and Lucy throws a temper tantrum. To calm her down, Susan relents and buys Lucy some candy. The next time they go shopping, Lucy sees candy and immediately throws another tantrum. This time, she obtains the candy quickly.
2. An interesting aspect of operant conditioning is that sometimes more than one actor is doing the learning. Scenario 1 is presented from Lucy’s point of view. But consider the same story from Susan’s point of view: Susan takes her toddler on a shopping trip. The child sees candy, wants it, and throws a tantrum. Overtired and in a rush, Susan gives the child some candy, and the tantrum stops. On the next trip, as soon as the child starts a preliminary wail, Susan quickly hands over some candy to stop the screaming.
3. Shevonne installs an electric fence system around the perimeter of her yard and gives her dog Snoopy a collar that makes a high-pitched noise whenever he gets too close to the boundary. The first time Snoopy strays out of bounds while wearing the collar, the noise plays and distresses him. Soon, Snoopy learns to avoid the noise by staying inside the yard.
4. Miguel’s football team has a no-alcohol policy: players sign pledges not to drink alcohol during the football season. One night, Miguel goes out with some friends and has a few beers. The coach finds out and revokes Miguel’s playing privileges for a week. When allowed to rejoin the team, Miguel is careful to stay away from alcohol for the rest of the season.
5. Rachel is a 10-year-old who hates gym class. One day, after eating the school lunch, she gets a stomachache. She

tells the school nurse she is feeling sick, and the nurse gives her a pass to skip gym class that afternoon. Now, on days when there is gym class, Rachel frequently feels sick after eating lunch at school.

Reinforcement Need Not Follow Every Response

In addition to controlling whether a response results in reinforcement or punishment being added or taken away, and whether the outcome ensues immediately or after a delay, an experimenter can also control the *frequency* with which these outcomes are delivered. The rules determining how and when outcomes are delivered in an experiment are called **reinforcement schedules**. So far, almost all of the examples in this chapter have been ones in which the outcome reliably follows the response. For instance, whenever the rat presses a lever, it gets food; whenever Becky sets the dinner table, she receives parental praise; and so on. These examples illustrate **continuous reinforcement schedules**, meaning that each response R is always followed by the outcome O.

reinforcement schedule

A schedule that determines how often reinforcement is delivered in an operant conditioning paradigm.

continuous reinforcement schedule

A reinforcement schedule in which every instance of the response is followed by the reinforcer.

But in some situations, a response must be repeated multiple times before triggering the expected outcome. For example, Becky has to clean her room seven days in a row to obtain her weekly allowance (seven responses for one reinforcement), and a baseball player is allowed to swing and miss three times before he strikes out (three responses for one punishment).

In the laboratory, an experimenter can devise a schedule defining exactly when outcomes are delivered. Patterns in which an outcome follows a response less than 100% of the time are called **partial reinforcement schedules** (or *intermittent reinforcement schedules*). The term *reinforcement schedule* is used here for simplicity, but these schedules can be applied either to reinforcement (Becky's weekly allowance) or to punishment (three strikes and you're out).



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What kind of learning is going on here?

partial reinforcement schedule

A reinforcement schedule in which only some instances of the response are followed by the reinforcer.

Reinforcement Schedules

There are four commonly used types of partial reinforcement schedules, defined by the specific rules determining when reinforcement arrives.

1. Fixed-ratio (FR) schedule. In an FR schedule, some fixed number of responses must be made before a reinforcer is delivered. For example, if a rat must press a lever five times to obtain one food pellet, the ratio of responses to reinforcers is 5:1; this is often called an FR 5 schedule. In this notation system, continuous reinforcement would be considered an FR 1 schedule: every (one) response results in reinforcement. The ratio can gradually be increased—for instance, by starting with an FR 1 schedule and working up through an FR 5 schedule to an FR 50 schedule and beyond—until the animal has been trained to make several hundred responses for each reinforcement on an FR schedule.

fixed-ratio (FR) schedule

A reinforcement schedule in which a specific number of responses must occur before a reinforcer is delivered; thus, FR 5 means that reinforcement arrives after every fifth response.

Rats on an FR schedule show a characteristic pattern of steady responding leading up to the reinforcement, followed by a few seconds with no responding. This short break in responding after a reinforcement is called the **post-reinforcement pause**. This pattern of responding under an FR schedule is most easily seen using a graph of cumulative responding (the type introduced in [Figure 5.2D](#)), which is one reason some researchers continue to display data in this format.

Figure 5.5A (blue line) shows the hypothetical behavior of a rat trained to respond on an FR 5 schedule: steady response rates leading up to each reinforcement (orange arrows), followed by a brief pause before another round of responding begins in order to obtain a new reinforcement.

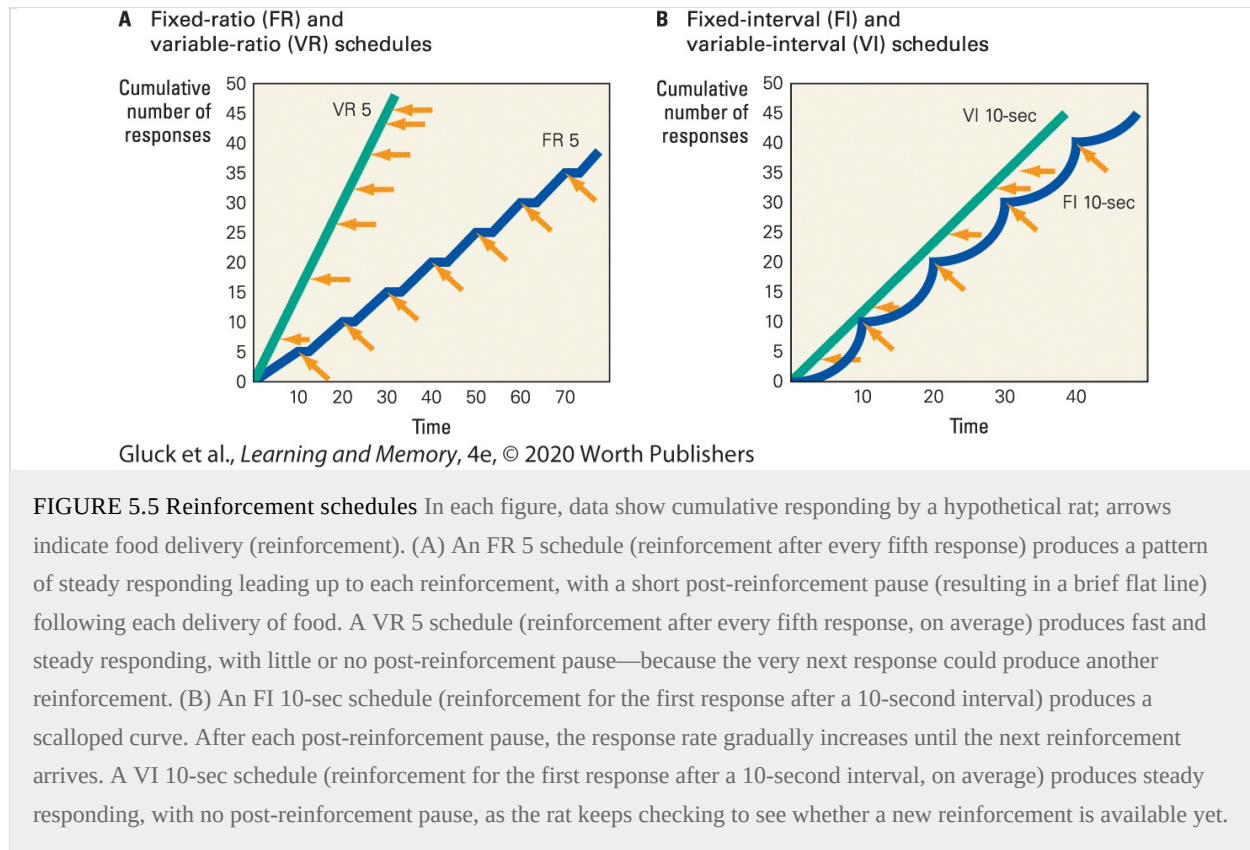


FIGURE 5.5 Reinforcement schedules In each figure, data show cumulative responding by a hypothetical rat; arrows indicate food delivery (reinforcement). (A) An FR 5 schedule (reinforcement after every fifth response) produces a pattern of steady responding leading up to each reinforcement, with a short post-reinforcement pause (resulting in a brief flat line) following each delivery of food. A VR 5 schedule (reinforcement after every fifth response, on average) produces fast and steady responding, with little or no post-reinforcement pause—because the very next response could produce another reinforcement. (B) An FI 10-sec schedule (reinforcement for the first response after a 10-second interval) produces a scalloped curve. After each post-reinforcement pause, the response rate gradually increases until the next reinforcement arrives. A VI 10-sec schedule (reinforcement for the first response after a 10-second interval, on average) produces steady responding, with no post-reinforcement pause, as the rat keeps checking to see whether a new reinforcement is available yet.

Description

The graph on the left shows Fixed-ratio (F R) and variable-ratio (V R) schedules. The X-axis shows Time, and the Y-axis shows the Cumulative number of response. The arrows indicate food delivery reinforcement. A F R 5 schedule reinforcement, after every fifth response, produces a pattern of steady response leading up to each reinforcement. A V R 5 schedule reinforcement after every fifth response, on average, produces fast and steady responding, with little or no post-reinforcement pause.

The graph on the right shows Fixed-interval (F I) and variable-interval (V I) schedules. The X-axis shows Time, and Y-axis shows the Cumulative number of response. An F I 10-second schedule, the reinforcement for the first response after a 10-second interval, generates a bead-like curve. After each post-reinforcement pause, the response rate steadily progresses until the next reinforcement arrives. A V I 10 second schedule, forms a straight line as the reinforcement for the first response after a 10-second interval, on average generates steady responding, with no post-reinforcement pause.

post-reinforcement pause

In operant conditioning, a brief pause in responding that follows delivery of the reinforcer.

During the post-reinforcement pause, it seems almost as if the animal is pausing to take a rest before its next bout of responding. And, in fact, the length of the post-reinforcement pause is related to the number of responses required to obtain the next reinforcement: thus, the post-reinforcement pause when an animal is on an FR 50 schedule is longer than when the animal is on an FR 5 schedule. In effect, the rat is behaving like a human teenager who completes a quick chore (say, taking out the trash) the first time his mother asks but procrastinates for hours before starting a really time-consuming chore (mowing the lawn).

Examples of fixed-ratio schedules in human life include factory workers getting paid a flat fee for every 100 pieces they turn out and farm workers getting paid a fixed amount for every bushel of apples picked. In fact, such workers tend to show behavior similar to that of rats on an FR schedule—steady bursts of responding followed by post-reinforcement pauses: the workers complete a batch, take a few minutes for a coffee break, and then start in again. A similar phenomenon occurs in readers: they may complete a chapter or a fixed number of pages before putting the book aside. Novelists often try to combat this “post-reinforcement pause” by ending each chapter with an exciting cliffhanger so that readers will read on to see what happens next.

2. Fixed-interval (FI) schedule. Whereas an FR schedule provides reinforcement after a fixed *number* of responses, an FI schedule reinforces the first response after a fixed *amount of time*. For example, on an FI 10-sec schedule, the rat is reinforced for the first response it makes after an interval of 10 seconds since the last reinforcement. Importantly, the reinforcement is not automatically obtained after the fixed interval but merely becomes available to be earned; the organism must still respond in order to actually receive that reinforcement. Once the interval has elapsed, the reinforcement remains available until the response occurs and the reinforcement is obtained. At that point, the clock starts ticking on the next fixed interval until the next reinforcement becomes available.

fixed-interval (FI) schedule

A reinforcement schedule in which the first response after a fixed amount of time is reinforced; thus, FI 1-min means that reinforcement follows the first response made after a 1-minute interval since the last reinforcement.

Under these circumstances, the most efficient strategy on the rat’s part would be to wait exactly 10 seconds after each reinforcement and then respond once to obtain the next reinforcement. Earlier responses (before the 10 seconds have elapsed) are wasted effort. However, [Figure 5.5B](#) shows how an animal on an FI schedule actually behaves: each reinforcement is followed by a

period of few or no responses, but the animal's rate of responding gradually increases as the end of the interval nears. Presumably, animals (including humans) cannot perfectly judge time intervals, so they estimate as best they can how much time has passed and err a little on the side of wishful thinking by responding too soon. The result is a characteristic “scalloped” cumulative response curve, as illustrated in [Figure 5.5B](#) (blue line).

An example of fixed-interval reinforcement would be a high school student being sentenced to serve detention from 3 p.m. to 4 p.m. After he arrives, there is little point in checking the clock for the first 15 or 20 minutes. But it might be worth checking after he estimates 30 to 40 minutes have passed, just in case time is going by faster than he thinks. As the elapsed time gets closer and closer to an hour, he might check the time more and more frequently, not wanting to stay a moment longer than necessary. In this example, the response is checking the time, the reinforcement is escape from detention, and only the first response after the end of the detention interval is reinforced—the rest are “wasted” responses. Notice that this is an FI schedule, not an FR schedule, because the rate at which the student checks his watch does not make the reinforcement appear any faster. Rather, the reinforcement arrives following the first response after the time interval has elapsed. Once 4 p.m. arrives, however, the student does not obtain reinforcement until he checks the clock and realizes he is free to go.



Ronnie Kaufman/The Image Bank/Getty Images

Some restaurants and stores provide “customer reward” cards (“Buy ten—get the eleventh one free!”), to modify customer behavior by increasing brand loyalty. Which type of reinforcement schedule does this represent?

3. Variable-ratio (VR) schedule. A VR schedule provides reinforcement after a certain average number of responses. For example, whereas an FR 5 schedule produces reinforcement after every fifth response, a VR 5 schedule produces reinforcement after every five responses *on average*. Thus, the responder never knows exactly when a reinforcement is coming. As a result, there is a steady, high rate of responding even immediately after a reinforcement is delivered because the very next response just might result in another reinforcement ([Figure 5.5A](#), green line). Thus, the VR schedule eliminates (or greatly reduces) the post-reinforcement pause observed under FR schedules.

variable-ratio (VR) schedule

A reinforcement schedule in which a specific number of responses, on average, must occur before a reinforcer is delivered; thus, VR 5 means that, on average, every fifth response is reinforced.

A real-life example of a VR schedule could be a slot machine. Even if you know that a particular

slot machine pays off on every 500th game, on average, you do not know exactly which games will pay off. Even if you have just hit the jackpot, the very next spin might be a winner, too, so there is a strong incentive to keep playing.

4. Variable-interval (VI) schedule. Whereas an FI schedule reinforces the first response after a particular time interval, a VI schedule reinforces the first response after an interval that averages a particular length of time. For example, a VI 10-sec schedule reinforces the first response after an interval that is 10 seconds *on average*—but the actual interval on any particular trial might be longer or shorter.

variable-interval (VI) schedule

A reinforcement schedule in which the first response after a fixed amount of time, on average, is reinforced; thus, VI 1-min means that reinforcement follows the first response made after a 1-minute interval, on average, since the last reinforcement.

In the VI schedule, as in the VR schedule, the responder never knows exactly when the next reinforcement is coming: responses made a few seconds after the previous reinforcement just might be reinforced, too. Thus, the response rate of animals under a VI schedule is usually steadier than under an FI schedule, as the animals check periodically to see whether reinforcement is available ([Figure 5.5B](#), green line).

An everyday example of the effect of a VI schedule is demonstrated by Tom, a college student whose girlfriend is studying abroad for a semester. She has promised to keep in touch through daily online posts, but sometimes she posts in the morning, sometimes in the evening—and sometimes more than once a day. Tom loves her and wants to read her posts as soon as they arrive, but he also does not want to spend 24 hours a day staring at his screen and waiting for the next post to come. A sensible compromise is for Tom to check online every few hours. This minimizes the time unread posts will sit waiting for him, while leaving him free to go pursue other interests in the meantime. Notice that this is a VI schedule, not a VR schedule, because the rate at which Tom checks online does not make the posts appear any faster. Once a new post does appear, it sits there waiting until Tom checks; his next response is rewarded, as he gets to read the post.

Just as VR schedules tend to produce higher rates of responding than FR schedules, so VI schedules tend to produce higher rates of responding than FI schedules. For example, if Tom's girlfriend posts every day at the fixed time of 2 p.m., Tom will tend to check online once every

day at about that time; if she posts at variable times, he is likely to check much more often, just in case a new post is waiting. One reason many people check their phones every few minutes is that texts and tweets arrive on a VI schedule: you can never be sure when there is a juicy new item sitting there, waiting to reinforce your behavior.

TEST YOUR KNOWLEDGE

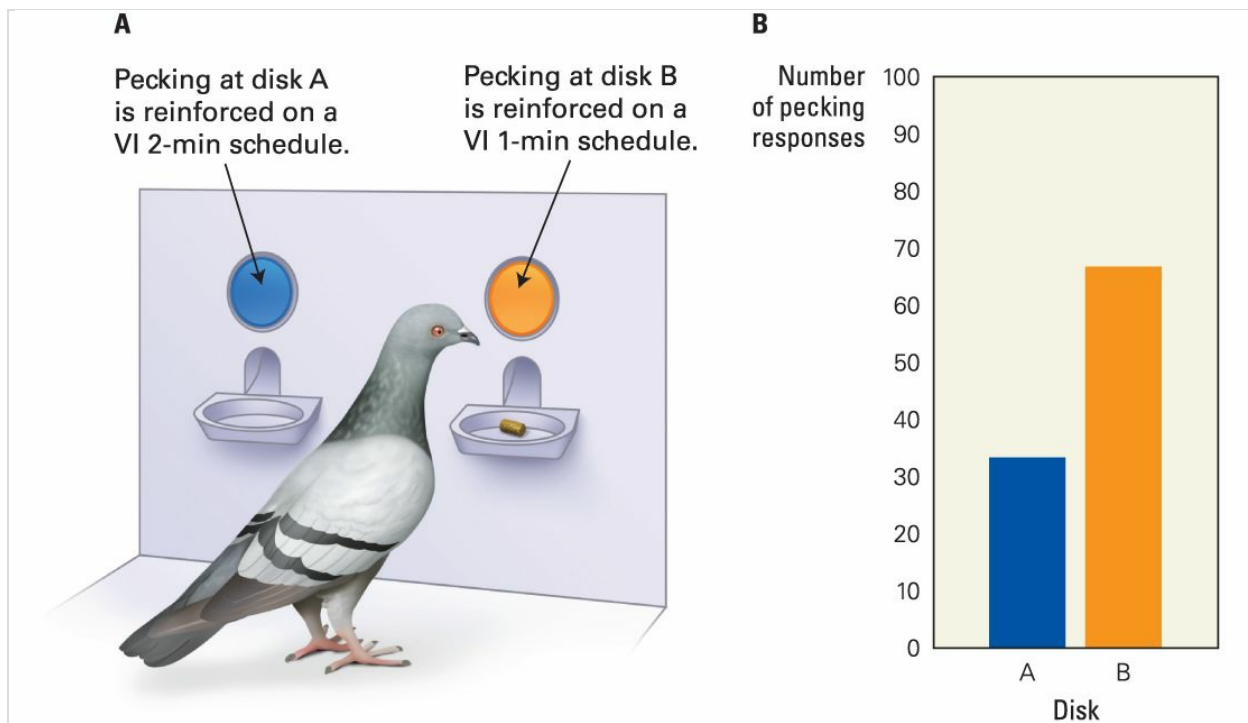
Reinforcement Schedules

Operant conditioning is common in human behavior. Try your hand at identifying whether each of the scenarios below is an example of an FR, FI, VR, or VI schedule. ([Answers appear at the end of the chapter.](#))

1. Each first-grade student who completes the day's math worksheet receives a gold star from the teacher; at the end of the week, five gold stars can be exchanged for a toy.
2. A good telemarketer scores an average of two sales for every 20 phone calls he makes, so he earns the most profit if he makes a lot of calls.
3. A couple go to their favorite restaurant on a Saturday night and are told that seating will be available in about 30 minutes. They wait at the bar and periodically return to the reception area to check whether a table is free.
4. Maria donates blood regularly at the local hospital; they pay her for her donations, and it makes her feel good to know she's helping people in need. However, due to hospital policy, donors have to wait at least 2 weeks between donations.
5. A surfer spends all available afternoons at his favorite beach, where he is sure of at least a couple of big waves every hour or so. After catching a big wave, he immediately paddles back out to await the next big one.
6. A man who likes to eat spicy foods for lunch always carries a pack of spearmint chewing gum so he can freshen his breath before returning to work in the afternoon.
7. A calculus teacher gives quizzes every Friday. Some students start looking over the material early in the week, but everyone in the class studies frantically on Thursday night. Usually, those who spend the most time studying get the best grades on the quiz.
8. A woman likes to play bingo at her local church. The game is set up so that 1 out of every 100 cards will be a winner, but it is impossible to know in advance which specific cards will win. To increase her chances of winning, the woman buys 10 cards each time she plays.

Choice Behavior

In addition to continuous and partial reinforcement schedules, there are also **concurrent reinforcement schedules**, in which the organism can choose between several possible responses, each leading to a different outcome. This allows researchers to examine *choice behavior*: how organisms choose to divide their time and efforts among different options. For example, suppose a pigeon is placed in a Skinner box with two illuminated disks, A and B (**Figure 5.6A**). Pecking at disk A is reinforced on a VI 2-min schedule (2-min = 2-minute), and pecking at disk B is reinforced on a VI 1-min schedule. What should the pigeon do?



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FIGURE 5.6 VI schedules and the matching law (A) A pigeon is presented with two illuminated disks; pecking at disk A is reinforced on a VI 2-min schedule, and pecking at disk B is reinforced on a VI 1-min schedule. The pigeon's optimal behavior would be to peck at B about twice as often as A—relative rates of responding that are proportional to the two different rates of reinforcement. (B) In fact, this is approximately what pigeons do: they spend about 67% (two-thirds) of their time pecking at disk B and about 33% of their time pecking at disk A.

Information from [Herrnstein, 1961](#).

Description

The illustration labeled A shows a pigeon kept in a position to peak two illuminated circular disks. The disc on the left reads, pecking at disk A is reinforced on a V I 2-minute schedule and the disc on the right reads, pecking at disk, B is reinforced on a V I 1-minute schedule.

The illustration labeled B shows a bar graph, with X-axis representing Disk and Y-axis representing Number of pecking responses. The data in the graph reads: The bar graph, with disk A has 33 percent of their time pecking, and disk B has 67 percent.

concurrent reinforcement schedule

A reinforcement schedule in which the organism can make any of several possible responses, each of which may lead to a different outcome (each of which may be reinforced according to a different reinforcement schedule).

Every 2 minutes, the pigeon can get one food pellet for pecking at disk A but gets two for pecking at disk B—so you might think that the pigeon would concentrate on pecking at B and

ignore A. But remember, if the experiment lasts longer than 2 minutes, a food pellet will be waiting to be delivered as soon as the pigeon pecks at A, and this pellet will never be obtained if the animal pecks only at B. The optimal behavior is some strategy that allows the pigeon to maximize the amount of food it can get from both disks, probably by spending the most effort pecking at B but occasionally switching over to peck at A, just to check. This would be analogous to channel surfing in humans: confronted with several possibly entertaining television programs, a typical response is to watch the preferred program most of the time but switch over to check out the other choices during the commercials, just in case something interesting is going on.

The Matching Law

Look more closely at the pigeon in [Figure 5.6A](#) and its choice between disk A on a VI 2-min schedule and disk B on a VI 1-min schedule. Can we be more precise about the pigeon's allocation of time? One way to discover the pigeon's strategy is simply to let the pigeon peck away for a few minutes, according to its own preference, and then calculate the proportion of time it spent pecking at each disk ([Figure 5.6B](#)). In fact, such calculations show that a pigeon will spend about 67% of its time pecking at B and about 33% of its time pecking at A—or about twice as many pecks at B as at A ([Herrnstein, 1961](#)). Note that this 2:1 ratio is identical to the relative rates of reinforcement on the two disks because B is reinforced twice as often as A. On the other hand, if A and B are reinforced equally often (say, both on a VI 1-min schedule), the pigeon will divide its time approximately equally between the two disks. This idea that an organism's response patterns will closely mimic the relative rates of reinforcement for each possible response is called the **matching law of choice behavior**.

matching law of choice behavior

The principle that an organism, given a choice between multiple responses, will make each response at a rate proportional to how often that response is reinforced relative to the other choices.

Of course, even within a lab experiment, a rat or pigeon has more options than just pecking at illuminated disks: it will spend some of its time eating the food it earns, and it can even take a break and spend some of its time grooming, exploring, or napping. Nevertheless, the matching law is a fairly good description of how the animal will allot its time and effort among a set of possible operant responses.

Behavioral Economics

A pigeon confronted with two disks is the simplest possible example of a choice situation. Outside the laboratory, choices are far more complicated. A college student has to divide her allotted studying time among different classes according to how that studying is likely to pay off best, and she has to divide her total time among studying, sleeping, eating, socializing, and so forth. A dieter who is allowed a fixed number of calories per day must decide whether to eat several low-calorie meals or splurge on a bowl of ice cream (and then survive on water and lettuce for the rest of the day).

Behavioral economics is the study of how organisms allocate their time and resources among possible options. For example, a worker who makes \$3,000 a month after taxes can distribute this income on rent, food, new clothes, savings, and so on. If she lives in a very expensive apartment, there is less money available for fancy food and new clothes; if she rents a less expensive apartment, she has more money to spend on other things. How does she choose?

behavioral economics

The study of how organisms allocate their time and resources among possible options.

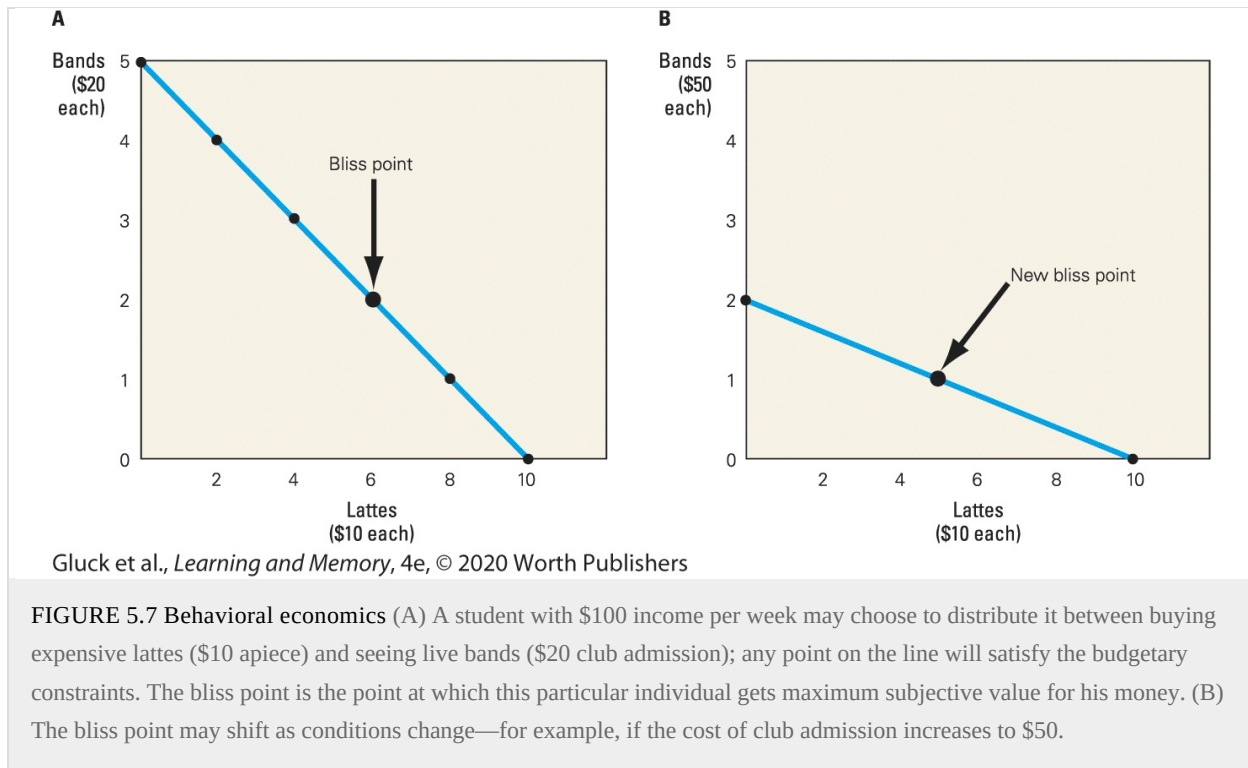
Economic theory predicts that each consumer will allocate resources in a way that delivers the maximum “subjective value,” or relative satisfaction, for that person. (In microeconomics, the word *utility* is used instead of subjective value.) The value is subjective because it differs from person to person: one individual may find much subjective value in an expensive apartment, but another may find more subjective value in having extra money to spend on clothes and food. The particular allocation of resources that provides maximal subjective value to an individual is called the **bliss point** ([Allison, 1983](#); [Timberlake, 1980](#)). We determine an individual’s bliss point simply by recording what that individual chooses to do.

bliss point

In behavioral economics, the allocation of resources that maximizes subjective value or satisfaction.

For example, consider Jamie, a college student with a part-time job that pays \$100 per week. Assuming that Jamie has no other expenses, he can spend this money to go see live music with his friends (say, \$20 admission at a local club) or splurge on his favorite morning coffee (say, \$10 for a large caramel latte with double whip from a trendy cafe). Each week he can spend the full \$100 to see 5 bands or buy 10 lattes or any other combination of expenditures that adds up to

\$100. [Figure 5.7A](#) shows Jamie's possible options; each point on the line is a different combination he can purchase with his \$100.



Description

The graph on the left plots bliss point of behavioral economics. The X-axis plots Lattes (10 dollars each), with values from 0 to 5 and Y-axis shows Bands (20 dollars each), with values from 0 to 10 in increments of 2. The line graph shows a straight line: 5 bands and no lattes, 4 bands and 2 lattes, and so on to 0 bands and 10 lattes. The bliss point is shown at 2 bands and 6 lattes.

The graph on the right plots new bliss point of behavioral economics. The X-axis plots Bands (50 dollars each), with values from 0 to 5 and Y-axis shows Lattes (10 dollars each), with values from 0 to 10 in increments of 2. The line shows a straight line: 2 bands and no lattes, 5 bands and 1 latte, and so on to 0 bands and 10 lattes. The bliss point is shown at 2 bands and 6 lattes.

Given these options, what does Jamie actually do? Suppose we record his behavior over the semester and find that, most weeks, he buys 6 lattes and sees 2 bands. This point (shown in [Figure 5.7A](#)) is Jamie's bliss point—the distribution of expenditures that (apparently) results in maximum subjective value for this individual: he gets his coffee most mornings, and he also gets to see plenty of live music. Of course, both the curve and the bliss point can shift if economic conditions change: if the club raises its prices (\$50 admission), then Jamie may shift to one

evening out and five lattes per week—resulting in the new bliss point shown in [Figure 5.7B](#).

Humans are not the only animals that have to choose how to allocate their time and energy among competing options. College student Jamie has a bliss point that reflects the monetary cost of his different options. Although animals do not use money, they also allocate their behaviors in a way that reflects the “cost” of each option. For example, predators such as sunfish must decide whether to invest energy chasing down a small prey animal like a guppy or wait for a larger one to come along—which could result in a bigger meal for the same energy expenditure. How to choose? One factor is the density of prey. If prey are rare, then the predator should probably chase any meal it sees, small or large. But if prey are plentiful, then the predator might as well skip the guppies and wait for a larger victim—which is just what the sunfish does. In a laboratory tank where large and small fish are plentiful, a sunfish generally only bothers to chase large prey. But when there are only a few fish in the tank, the sunfish will go after both large and small prey—whatever it can find ([Warner & Hall, 1974](#)). The sunfish’s bliss point—the allocation of its resources among different classes of prey—changes when environmental conditions change, just as Jamie’s bliss point shifts when economic conditions change.

Similarly, a naive rat in a Skinner box will spend some amount of time grooming, exploring, and sitting quietly—and a very small amount of time pressing the lever. Now, assume each lever press results in delivery of a single pellet of rat chow on an FR 1 schedule. As the rat learns this association (and assuming that the rat is hungry), it will begin to spend more of its time on lever-pressing (and eating) and proportionately less time on other activities because the subjective value of pressing the lever has been increased. If a second lever is added, and if pressing that new lever results in a preferred food (sugar pellets) on an FR 1 schedule, the animal will tend to press the new lever more often than the old. But if the “cost” of obtaining sugar pellets is then increased, say from an FR 1 schedule to an FR 10 schedule, the rat may switch back to the less preferred but more easily obtainable rat chow—analogous to Jamie’s decision to see live music less often if the cost of admission increases. From a behavioral economics perspective, then, operant conditioning is not so much training an organism to execute a specific behavior as causing the organism to shift its allocation of time and energy among possible behaviors ([Baum, 2002, 2004](#)).

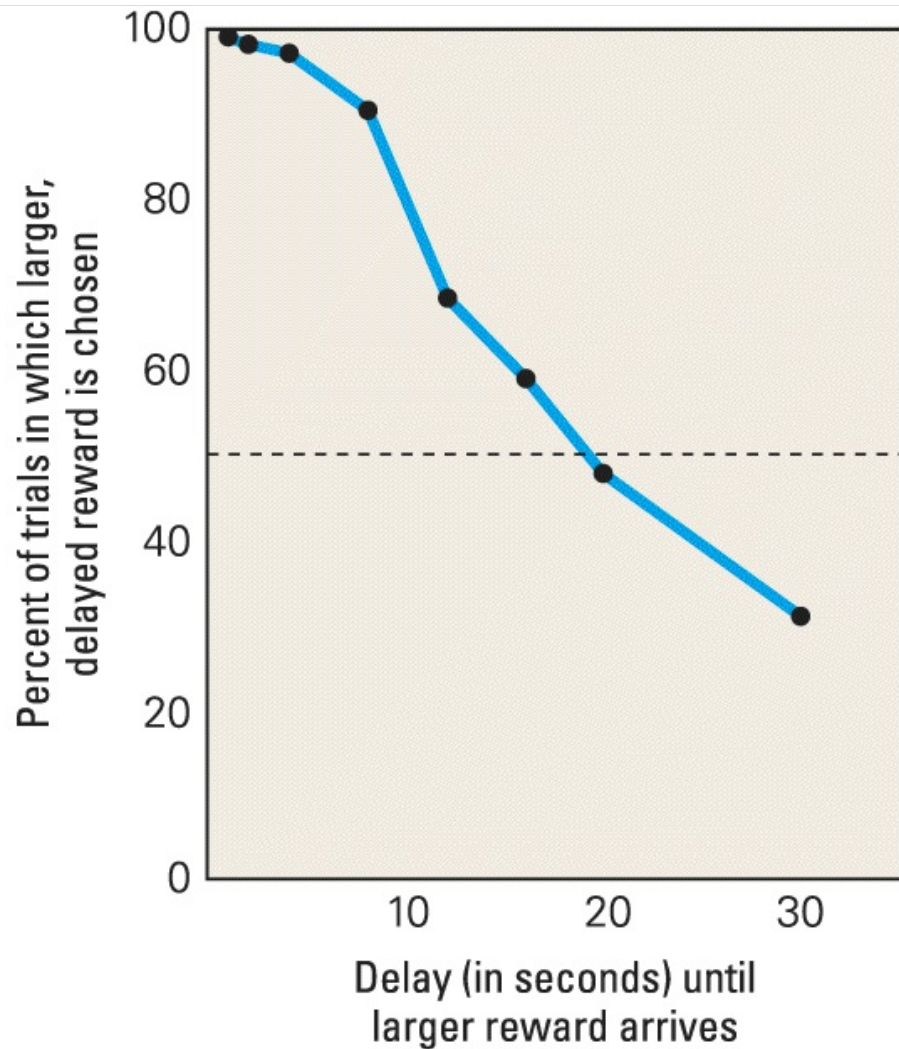
Delayed Reinforcement and Self-Control

Another factor that influences how organisms allocate their time and resources is the expected time until the outcome arrives. For example, given a choice between two levers, one that produces a large reward (say, 3 food pellets) and one that produces a small reward (say, 1 pellet), rats reliably respond by pressing on the high-payoff lever. Similarly, given a choice between an envelope containing \$500 and an envelope containing \$1,000, you would probably choose the larger sum. But what if the choice were between \$500 right now and \$1,000 mailed to you in 1 year? This choice would be more difficult because—for most people—the subjective value of a reward is progressively reduced (or discounted) the longer that reward is delayed. This phenomenon is known as [delay discounting](#). Put simply, a bird in the hand is worth two in the bush.

delay discounting

The progressive reduction (or discounting) of the subjective value of a reward the longer it is delayed.

For instance, suppose a rat is trained in a Skinner box with two levers: pressing one lever results in delivery of a small, immediate food reinforcement (1 food pellet), whereas pressing on the other lever results in a larger food reinforcement (3 pellets) that arrives after some delay. As shown in [Figure 5.8](#), if the delay is short (e.g., 1 or 2 seconds), a rat will choose the larger reward nearly 100% of the time. But as the delay gets progressively longer, the rat becomes less willing to wait, opting more and more often for the smaller immediate reward (e.g., [Mobini et al., 2002](#)).



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FIGURE 5.8 Delay discounting Rats were given a choice between two levers: one that delivers a small, immediate reward (1 food pellet) and one that delivers a larger reward (3 pellets) after some delay. If that delay was short (e.g., 0 to 5 seconds), rats would choose the larger reward on nearly 100% of choice trials. But as the delay grew longer (e.g., 20 to 30 seconds), they were increasingly less likely to wait, opting more often for the smaller, immediate reward.

Information from [Mobini et al., 2002](#).

Description

The X-axis plots Percent of trails in which larger, delayed reward is chosen, and Y-axis shows Delay (in seconds) until larger reward arrives. The data in the graph form a negative curve and the points plotted are as follows: (30, 38), (20, 49), (15, 60), (10, 70), (5, 98), (3, 99), and (1,100). All the data are approximate.

The same trade-off occurs in humans: it is easy to convince a student to put off fun in order to study if the exam is coming up tomorrow; it is harder if the exam is not for 5 weeks, even though starting to study early will result in a better grade. The delay between response (early start to studying) and reinforcement (good grade) makes the reinforcement less effective in evoking the response (as demonstrated in [Figure 5.4](#)). Similarly, one reason a weight-loss diet is difficult to maintain is that, at each meal, the dieter has to choose between the immediate reward of a dessert and the delayed reward of future weight loss.

Self-control refers to an organism's willingness to forgo a small immediate reward in favor of a larger future reward. The degree of self-control differs across individuals and across the age span. For example, when asked, "Would you rather receive \$500 today or \$1,000 in 1 year?" adults in their 60s are highly likely to choose the larger, delayed reward; college students are somewhat less likely to choose the delayed reward; and 12-year-olds almost never choose the delayed reward—preferring immediate gratification ([Green, Fry, & Myerson, 1994](#)).

self-control

An organism's willingness to forgo a small immediate reward in favor of a larger future reward.

One way of improving an individual's ability to wait for a reward is to induce the person to make a *precommitment*—that is, to make a choice that is difficult to change later. So, for instance, a student may be more likely to study early in the semester if she joins a weekly study group, in which case she will experience peer pressure to attend the group and to study a little each week. A dieter may be less likely to cheat on his diet if he first empties the kitchen of chips and ice cream, so that when the cravings hit, it will be difficult to sneak some junk food. These precommitments do not make it impossible to get the immediate reward (the student can skip a study meeting, and the dieter can drive to the supermarket to buy ice cream), but they do make it harder to get the immediate reward, and the individual is consequently more likely to stick by an earlier decision in favor of the later, larger reward.

Reciprocity and Altruism

As you have now seen, many seemingly complex choice behaviors can be explained in terms of operant conditioning, involving an $S^D \rightarrow R \rightarrow O$ association. You have learned that people and other animals will choose among possible behaviors in a way that maximizes expected subjective value and that they can show self-control, or the willingness to work now for anticipated future

rewards. These behaviors all seem generally consistent with Thorndike's law of effect (described earlier), which states that actions resulting in desirable outcomes (reinforcers) should increase in frequency and that actions resulting in undesirable outcomes (punishers) typically decrease in frequency.

But there are other behaviors that seem to violate the law of effect. An important example is [altruism](#), which occurs when organisms act in ways that benefit another—at the expense of some cost to themselves. Examples of altruism in human behavior would be an individual donating food or money to help victims of a natural disaster and someone waiting in line allowing another to go first.

altruism

In behavioral economics, an action or a behavior that provides benefit to another at the expense of some cost to the actor.

Other animals appear to show altruistic behavior as well. Among social insects such as some bees and wasps, individuals may sacrifice their own lives to care for and defend the queen and her offspring in the hive ([Gadagkar, 2016](#); [Nouvian, Reinhard, & Giurfa, 2016](#)). Among meerkats, a male will “volunteer” to babysit for the day, sacrificing his own opportunity to eat, while others in the group forage for food ([Carlson et al., 2006](#)). And dolphins have been observed swimming for hours underneath an injured female dolphin, helping to support her body near the surface so she can continue breathing ([Park et al., 2013](#)). These seemingly altruistic actions benefit the recipients—but carry high costs to the actors. Yet the principles of operant conditioning predict that if an action results in punishment, such as loss of time and resources, then the frequency of that action should decrease, and altruistic behaviors should extinguish. Why, then, do these altruistic behaviors persist?

Many apparently altruistic acts occur in the context of kin relationships, as when parents sacrifice time and energy to feed and care for their young, sometimes risking death or injury to protect the young from predators. Among many social animals, such as meerkats and social insects, most of the individuals in a colony are closely related—an extended “family.” In these cases, animals performing altruistic acts sacrifice their own well-being to promote the welfare of their kin. This has been explained in terms of natural selection: an individual's kin (children, siblings, nieces, and nephews) share many of the same genes, so that acting to help those kin survive into the next generation is actually a way to promote the survival of one's own genes ([Hamilton, 1963](#)). Thus,

genes that predispose animals to behave in seemingly altruistic ways toward their kin are genes that are likely to be passed down to future generations, which will in turn also tend to behave in altruistic ways. Much seemingly altruistic behavior among kinship groups may be interpreted this way ([Griffin & West, 2003](#)).

However, not all altruistic behavior is easily explained in terms of promoting kin survival. Altruistic acts sometimes occur between “friends” who are not genetically related. For example, genetically unrelated macaque monkeys will sit and groom one another, combing through each other’s fur to find and remove small parasites ([Adiseshan, Adiseshan, & Isbell, 2011](#)); unrelated male dolphins may form “alliances” to help guard each other’s female consorts ([Connor, 2010](#)); and red-winged blackbird males will help to defend unrelated neighbors’ nests ([Olendorf, Getty, & Scribner, 2004](#)). Why would animals behave this way?

One possible explanation for these actions is the principle of **reciprocal altruism**, which states that an organism may donate time or resources to help another in the expectation that the individual will return the favor later on ([Trivers, 1971](#)). This is similar to the concept of “tit-for-tat” behavior, which can be roughly restated as “you scratch my back, I’ll scratch yours” ([Axelrod & Hamilton, 1981](#)). For example, monkey A grooms monkey B now, in expectation that B will reciprocate later on. If that expectation pays off, it provides delayed reinforcement for monkey A’s original act, which in turn increases the likelihood that A will groom B again in the future. Similarly, humans who lend money or emotional support to a friend may do so in the expectation that someday they themselves might need help, and the friend will return the favor.

reciprocal altruism

In behavioral economics, the principle that one organism may donate time or resources to help another in the expectation that the other will return the favor later on.

In this way, many acts of friendship and cooperation among humans and other animals can be interpreted in the context of operant conditioning:

S^D (friend needs favor) → R (perform favor now) → O (receive future benefits)

Consistent with this interpretation, male red-winged blackbirds will stop defending their neighbors’ nests if those neighbors do not reciprocate ([Olendorf et al., 2004](#)), and humans will typically stop behaving generously to friends who fail to reciprocate in some fashion to sustain

the friendship. Across a range of species, animals seem to be able to keep track of services given and received over considerable periods of time, and animals that have recently received small acts of “friendship,” such as co-grooming, may be more likely to offer more valuable services, such as defense, in the future ([Seyfarth & Cheney, 2012](#)).

But there are other examples of altruism that do not obviously involve expectations of reciprocity, as when a donor gives money to a charity that will redistribute the money to needy individuals who will never know the identity of the donor—and who are thus unable ever to reciprocate directly. Some humans even donate organs to strangers they will never meet, at potential cost to their own health and fitness. Even in the laboratory, when participants are asked to allocate resources between themselves and an anonymous partner, some participants (but not all!) choose to behave altruistically even though they will never meet the partner again ([Seymour, Yoshida, & Dolan, 2009](#)).

These selfless acts, undertaken without expectation of direct repayment, are harder to explain in the context of the law of effect. Possibly, the actor receives reinforcement in the form of societal approval of the good deeds, or even an internal feeling of “doing good” that reinforces the altruistic behavior. More recently, researchers have proposed that, at least for animals in social groups, the simple rule of “tit-for-tat” can be expanded to a more general rule of “help anyone if helped by someone” ([Taborsky, Frommen, & Riehl, 2016](#)). For instance, a male meerkat may volunteer to babysit today, protecting his own offspring along with the rest of the infants, in the general expectation that other males in the group will take their turns on subsequent days. Similarly, a person may help a stranger today in expectation that, in the future, someone else (or society at large) will come to his aid in return.

Of course, operant conditioning cannot easily explain all instances of altruism; in humans, cultural, philosophical, and religious factors also play a role. Nevertheless, altruism is an example of a very complex behavioral phenomenon that can at least partially be interpreted via the principles of operant conditioning and delayed reinforcement.

[Interim Summary](#)

- In operant conditioning, organisms learn to make responses under particular conditions in order to obtain or avoid outcomes: Discriminative stimulus $S^D \rightarrow$ Response $R \rightarrow$ Outcome

O.

- In operant conditioning, the outcome (reinforcement or punishment) occurs only if the organism makes the response. In classical conditioning, by contrast, the unconditioned stimulus (US) occurs whether or not the organism makes a conditioned response (CR).
- Discriminative stimuli signal to the organism whether a particular response will result in a particular outcome.
- A reinforcer is an outcome that an organism will work to obtain; a punisher is an outcome that an organism will work to avoid. While punishment can be effective in eliminating an undesired response, it leads to more varied behavior and can be undermined by discriminative stimuli that encourage cheating, by concurrent reinforcement, or by weakness of the initial punisher. Another approach to eliminating unwanted behavior is differential reinforcement of alternative behaviors (DRA).
- Complex responses can be trained by shaping, in which progressive approximations to the desired response are reinforced, as well as by chaining, in which organisms are gradually trained to execute a sequence of responses.
- The four basic types of operant paradigm are positive reinforcement, negative reinforcement, positive punishment, and negative punishment. The words *positive* and *negative* denote whether the outcome is added to or subtracted from the environment; *reinforcement* and *punishment* denote whether the response increases or decreases in frequency as a result of learning.
- Schedules of reinforcement define whether the outcome O follows every response R, is available after some (fixed or variable) number of responses, or is available only after some (fixed or variable) time interval.
- When different responses are reinforced under VI schedules, the matching law predicts that organisms will allocate time among those responses based on the relative rates of reinforcement for each response.
- Behavioral economics is the study of how organisms choose to allocate their time and resources among various responses that result in different outcomes. The bliss point is the particular allocation of resources that provides maximal subjective value to an individual.
- Altruism refers to actions that are costly to the actor but that benefit another. Altruism toward one's kin can partially be explained in terms of natural selection. Altruism among "friends" may partially be explained as operant conditioning in terms of a tit-for-tat model.

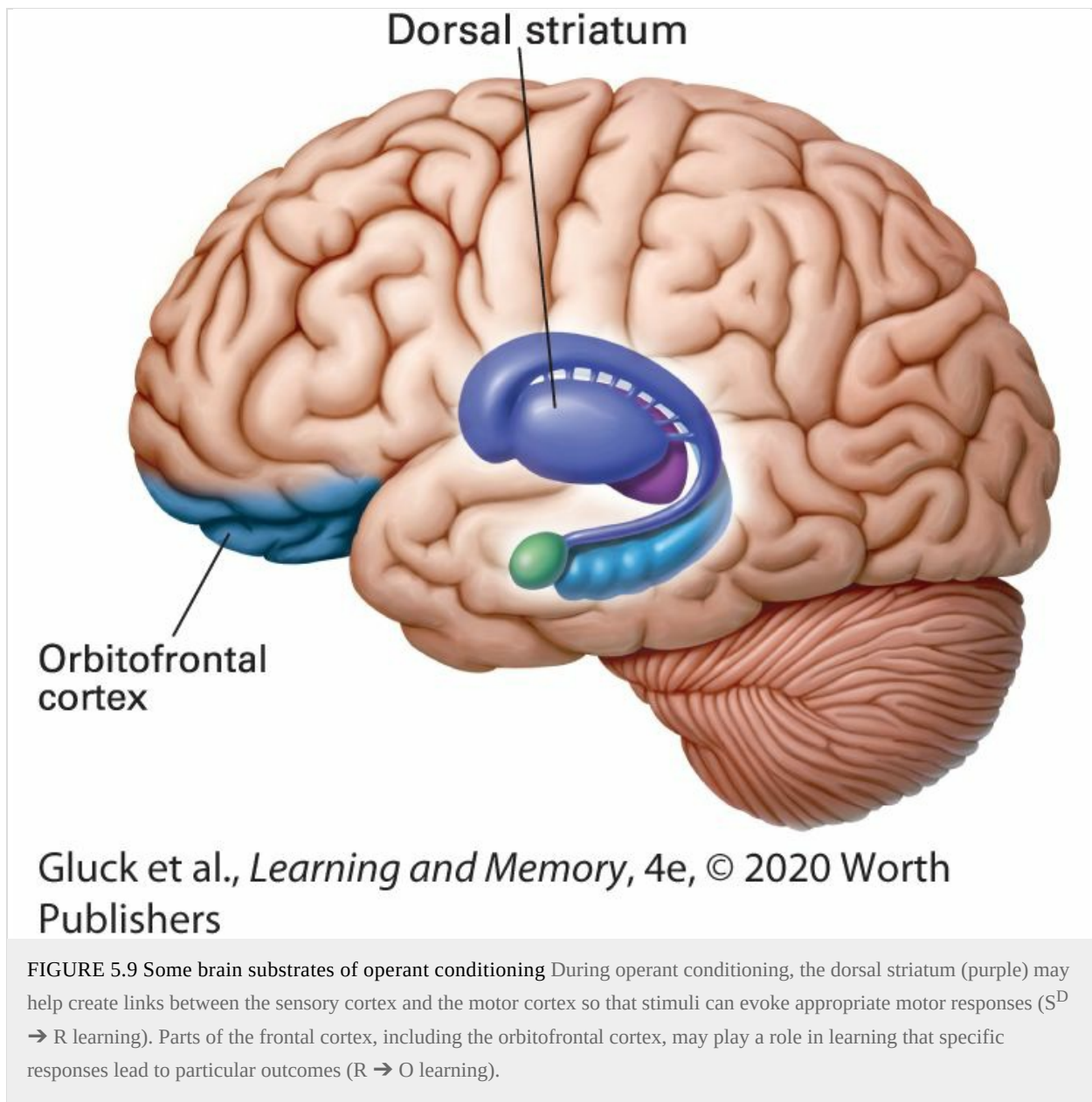
5.2 Brain Substrates

The previous section defines *operant conditioning* as learning an $S^D \rightarrow R \rightarrow O$ association. In studying such associations, neuroscientists are discovering that the parts of the brain that link stimuli with responses ($S^D \rightarrow R$ learning) are different from the parts of the brain that learn about the expected outcomes of those responses ($R \rightarrow O$). While many brain areas play roles in these processes, two key areas are the dorsal striatum, which appears to be particularly important for $S^D \rightarrow R$ learning, and the orbitofrontal cortex, which appears to be important for learning about expected outcomes. Still other brain areas may help us evaluate whether those outcomes are reinforcers or punishers.

The Dorsal Striatum and Stimulus–Response ($S^D \rightarrow R$) Learning

Voluntary motor responses occur when neurons in the motor cortex send messages to motor neurons in the muscles that control movements. The motor cortex receives its primary inputs from cortical areas that process sensory information, such as the visual cortex (V1) and the somatosensory cortex (S1), which you saw back in [Figure 2.8](#). It also receives inputs from the frontal cortex. When you see a book, this visual stimulus is registered by your visual cortex. If you decide to pick up the book, this “decision” is made in your frontal cortex. Signals from both the visual cortex and the frontal cortex then travel to your motor cortex, which integrates these signals and produces the appropriate instructions, causing your arm and hand to move in a coordinated way as you pick up the book.

Information from the sensory cortex to the motor cortex can also travel by an indirect route, through the [basal ganglia](#). The basal ganglia are a collection of *ganglia*—that is, clusters of neurons—that lie under the cortex, on either side of the thalamus. One part of the basal ganglia is the [dorsal striatum](#), shown in [Figure 5.9](#), which can be further subdivided into the *caudate nucleus* and the *putamen*. The dorsal striatum receives highly processed stimulus information from the sensory cortex and projects it to the motor cortex, which produces a behavioral response.



basal ganglia

A brain region that lies at the base of the forebrain and includes the dorsal striatum.

dorsal striatum

A region of the basal ganglia that is important for stimulus–response learning.

The dorsal striatum plays a critical role in operant conditioning, specifically the ability to learn $S^D \rightarrow R$ associations based on feedback about reinforcement and punishment ([McDonald & White, 1994](#); [O’Doherty et al., 2004](#)). Rats with lesions of the dorsal striatum can learn simple operant responses (e.g., when placed in a Skinner box, they can learn to lever-press R to obtain

food O). But if discriminative stimuli are added (e.g., if lever-press R is reinforced only in the presence of a light S^D), then the lesioned rats are markedly impaired ([Featherstone & McDonald, 2004](#)). In humans, too, individuals with damage or disruption to the striatum due to Parkinson's disease or Huntington's disease show deficits in the ability to associate a discriminative stimulus with a correct response ([Ashby & Waldron, 2000](#); [Robbins, 1996](#)).

$S^D \rightarrow R$ associations that depend on the dorsal striatum tend to be relatively automatic or habitual ([Balleine, Daw, & O'Doherty, 2008](#)). Remember the well-trained rats, discussed earlier in the chapter, that would run right through a pile of food on their way to a goal box in the maze? That behavior probably reflects $S^D \rightarrow R$ learning in the striatum, making the maze-running automatic even when other behaviors (such as pausing to eat) would have resulted in reward. In this case, running is based on a history of learning that the response resulted in desirable outcomes; but after a long period of training, the response is performed even though the outcome is no longer contingent on that action.

The Orbitofrontal Cortex and Learning to Predict Outcomes

$S^D \rightarrow R$ learning is, of course, only part of the picture in operant conditioning. Organisms learn to predict that particular responses R (in the presence of S^D) will result in particular outcomes O. For example, in [Section 5.1](#), you read about the negative contrast effect: monkeys may shriek in annoyance if their response earns them a less preferred food than the one they expected, and trick-or-treaters may feel cheated if they receive raisins rather than the expected candy. Such results show that organisms make responses in anticipation of particular outcomes.

Several brain areas appear to be involved in learning to predict the outcomes of behavior. Among these are parts of the *prefrontal cortex*, including the **orbitofrontal cortex**, which lies at the underside of the front of the brain in primates (see [Figure 5.9](#)) and which appears to contribute to goal-directed behavior by representing predicted outcomes ([Schoenbaum, Roesch, Stalnaker, & Takahashi, 2009](#); [Tanaka, Balleine, & O'Doherty, 2008](#)). The orbitofrontal cortex receives inputs that convey the full range of sensory modalities (sight, touch, sound, etc.), as well as visceral sensations (including hunger and thirst), allowing this brain area to integrate many types of information. Outputs from the orbitofrontal cortex travel to brain areas, including the striatum,

where they can help determine which motor responses are executed.

orbitofrontal cortex

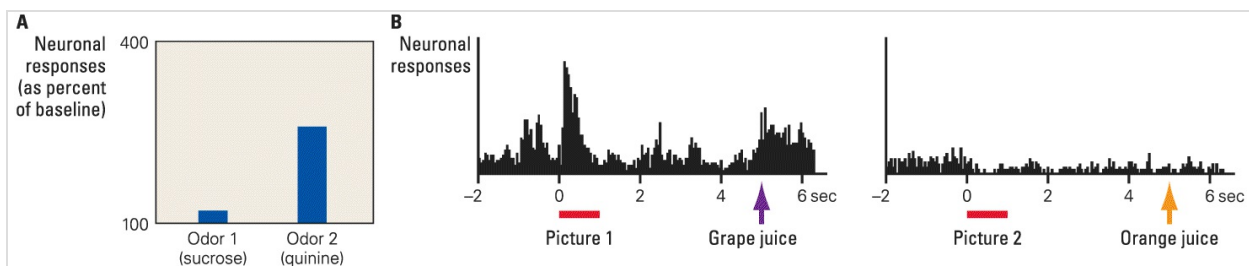
A brain region that is important for learning to predict the outcomes of particular responses.

Predicting Specific Outcomes

Some of the strongest evidence that the orbitofrontal cortex plays a role in predicting the outcomes of responses comes from neuronal recordings. For example, thirsty rats can be trained on a discrimination task in which the discriminative stimuli are two odors, the response R is to poke the nose into a nearby water cup, and the two possible outcomes are a tasty sucrose solution or a bitter quinine solution. A short delay (typically less than a second) is introduced between the response and the outcome, during which period a trained animal is “expecting” the outcome.

During this delay, some neurons in the orbitofrontal cortex fire differently, depending on whether the animal expects a reward or a punisher ([Schoenbaum, Chiba, & Gallagher, 1998](#)).

[Figure 5.10A](#) illustrates the firing patterns of one neuron in the orbitofrontal cortex of a rat learning such a task. This particular neuron fires strongly if the rat has just made a (mistaken) response to odor 2 and is expecting quinine, but it fires less strongly if the rat has just made a response to odor 1 and is expecting sucrose. Therefore, this neuron appears to code expectation of the punisher rather than the reward. If the contingencies are reversed, such that odor 1 now predicts quinine and odor 2 now predicts sucrose, those neurons often alter their responses to reflect the new contingencies ([Stalnaker, Franz, Singh, & Schoenbaum, 2007](#)).



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FIGURE 5.10 Orbitofrontal neurons code expected outcomes (A) Responses of a single neuron in the orbitofrontal cortex of a rat trained to expect that responding to odor 1 produces sucrose while responding to odor 2 produces quinine. The neuron is observed after the rat has made its behavioral response but before the outcome (sucrose or quinine) is delivered. This neuron shows very little increase above its baseline activity (100%) after the rat has responded to odor 1 but displays heightened activity after the rat has (mistakenly) responded to odor 2. Thus, this neuron appears to code for expectation of quinine. (B) Activity of a single neuron in the orbitofrontal cortex of a monkey trained to expect that some pictures (e.g., picture 1) predict that responding will be followed by grape juice, but other pictures (e.g., picture 2) predict that responding will be followed by orange juice. Each picture is presented for 1 second, as indicated by the red bars, with juice delivered 4

seconds later, as indicated by arrows. This neuron responds strongly (height of bars indicate neuronal activity) when picture 1 (which predicts grape juice) appears and again when the grape juice is delivered. But the same neuron does not fire to picture 2 (which predicts orange juice) nor when the orange juice is delivered. Thus, this neuron appears to code expectation of a specific outcome: grape juice.

(A) Data from [Schoenbaum et al., 1998](#), Figure 3a. (B, C) Information from [Tremblay & Schultz, 1999](#), Figure 3b.

Description

A bar graph labeled A plots responses of a single neuron in the orbitofrontal cortex of a rat. The X-axis shows neuronal responses (as percent of baseline) and Y-axis shows Odor 1 (sucrose) and odor 2 (quinine). Odor 2 (quinine) is higher than Odor 1 (sucrose) which is just above its baseline activity (100 percent).

A graph labeled B shows the activity of a single neuron in the orbitofrontal cortex of a monkey. The graph on the left shows neuronal responses with Picture 1, followed by Grape juice and the graph on the right shows neuronal responses with Picture 2 and Orange juice. The peaks on Picture 1 and Grape juice are higher than Picture 2 and Orange juice.

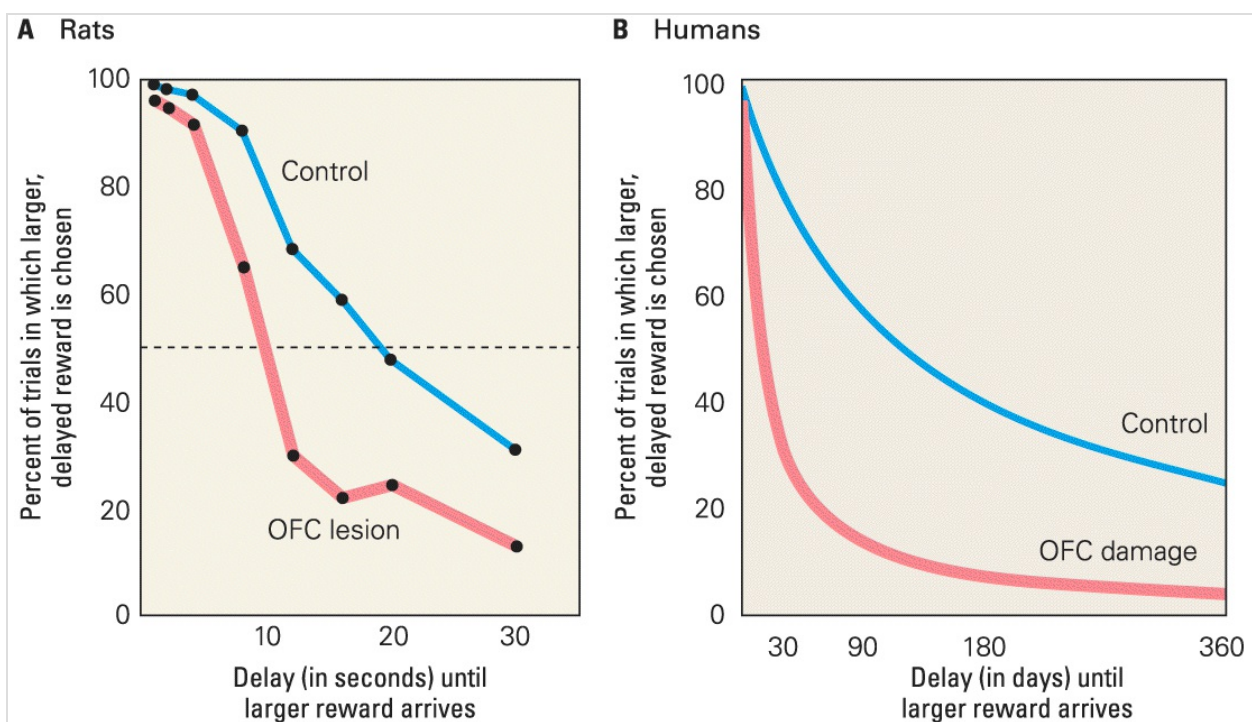
Neurons in the orbitofrontal cortex do not merely learn whether to expect reinforcement or punishment; they apparently even code the actual identity of the expected outcome. Thus, monkeys can be trained to differentiate a set of pictures that predict whether the upcoming reward will be grape juice or orange juice. [Figure 5.10B](#) shows the responses of a single neuron that became active whenever a stimulus that predicted grape juice was presented but not when pictures predicting orange juice were presented ([Tremblay & Schultz, 1999](#)). This same neuron also fired during the actual delivery of grape juice but *not* during the delivery of orange juice.

Given their ability to encode specific predicted outcomes, orbitofrontal cortex neurons play an important role in helping us select between potential actions based on their expected consequences. Recall college student Jamie, who could spend his weekly income by distributing it among choices such as music and lattes. Possibly, neurons in Jamie's orbitofrontal cortex were helping him evaluate the potential outcomes of his actions so that he could act to maximize the expected subjective value.

Delayed Reinforcement and Self-Control

In addition to processing the identity of expected rewards, the orbitofrontal cortex appears to evaluate the expected timing of those rewards. Remember the rats in [Figure 5.8](#) that could choose between pressing one lever to obtain a small immediate food reward or pressing another lever for a larger but delayed reward? The data are replotted in [Figure 5.11A](#) ("control"). As you saw before, if the larger reward is due after a short delay (e.g., less than 2 seconds), healthy

(control) rats will almost always choose to wait for that larger reward; but as the delay for the larger reward increases (e.g., 30 seconds), the rats become increasingly more likely to choose the smaller, immediate reward. Presumably, the rats are making an internal calculation of subjective value, comparing the two expected outcomes. The orbitofrontal cortex appears to be important for making these assessments of subjective value. [Figure 5.11A](#) (“OFC lesion”) shows that rats with lesions of the orbitofrontal cortex will still choose the delayed reward if the delay is short; however, as the delay increases, their willingness to wait plummets ([Mobini et al., 2002](#)). In effect, these lesioned rats show decreased self-control—less willingness to work for an expected future payoff.



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FIGURE 5.11 Orbitofrontal cortex and self-control (A) If control rats are given a choice between an immediate small reward and a larger, delayed reward, they will almost always prefer the larger reward if the delay is short (e.g., a few seconds), but as the delay increases, they will increasingly prefer the immediate reward. Rats with lesions of the orbitofrontal cortex (OFC) show much less self-control, indicated by a greatly decreased willingness to wait for the larger, delayed reward. (B) Humans show a similar pattern: at short delays, both healthy (control) participants and people with OFC damage will usually opt for the larger reward; but as the delay increases, the people with OFC damage show much less self-control, indicated by decreased willingness to wait for the larger, delayed reward.

(A) Data from [Mobini et al., 2002](#); (B) Data from [Sellitto et al., 2010](#), Figure 3 –left.

Description

The graph labeled A shows orbitofrontal cortex and self-control in rats. The X-axis marks Percent of trials in which larger, delayed reward is chosen, and Y-axis marks Delay in seconds until larger reward arrives. If control rats are given a choice between an immediate small reward and a larger, delayed reward, they prefer the larger reward if the delay is short. As the delay increases, the rats with O F C lesion frequently prefer the immediate reward.

The graph labeled B shows orbitofrontal cortex and self-control in human. The X-axis marks Percent of trials in which larger, delayed reward is chosen, and Y-axis marks Delay in seconds until larger reward arrives. The data in the graph is as follows: Two curves, Control, and O F C damage, are shown.

People with O F C damage opt for the larger reward. But as the delay increases, it shows much less self-control, indicated by decreased readiness to wait for the larger, delayed reward.

Humans show a similar effect, as illustrated in [Figure 5.11B](#). Healthy humans, given a choice between a small immediate reward (say, \$20 now) or a larger reward after a short delay (say, \$40 in 30 days), will usually prefer to wait for the larger reward; but as the delay increases, they become increasingly likely to prefer the smaller immediate reward. People with damage to the orbitofrontal cortex also prefer the larger reward if the delay is short; but as the delay increases, their ability to delay gratification plummets ([Sellitto, Ciaramelli, & de Pellegrino, 2010](#)). In effect, they become like college students who will study the night before an exam but are unwilling to start studying a month or a week earlier in anticipation that the effort now will pay off later.

Mechanisms of Reinforcement Signaling in the Brain

Operant conditioning involves learning that responses (R) in the presence of certain stimuli (S^D) will result in particular outcomes (O). You have just read that neurons in the orbitofrontal cortex help encode the identity of specific outcomes (e.g., grape juice versus orange juice). If an outcome is reinforcing, the $S^D \rightarrow R$ association should be strengthened, increasing the likelihood that S^D evokes R in the future; if it is a punisher, the association should be weakened, decreasing the likelihood of R. But how does the brain determine whether an outcome should be interpreted as a reinforcer or a punisher?

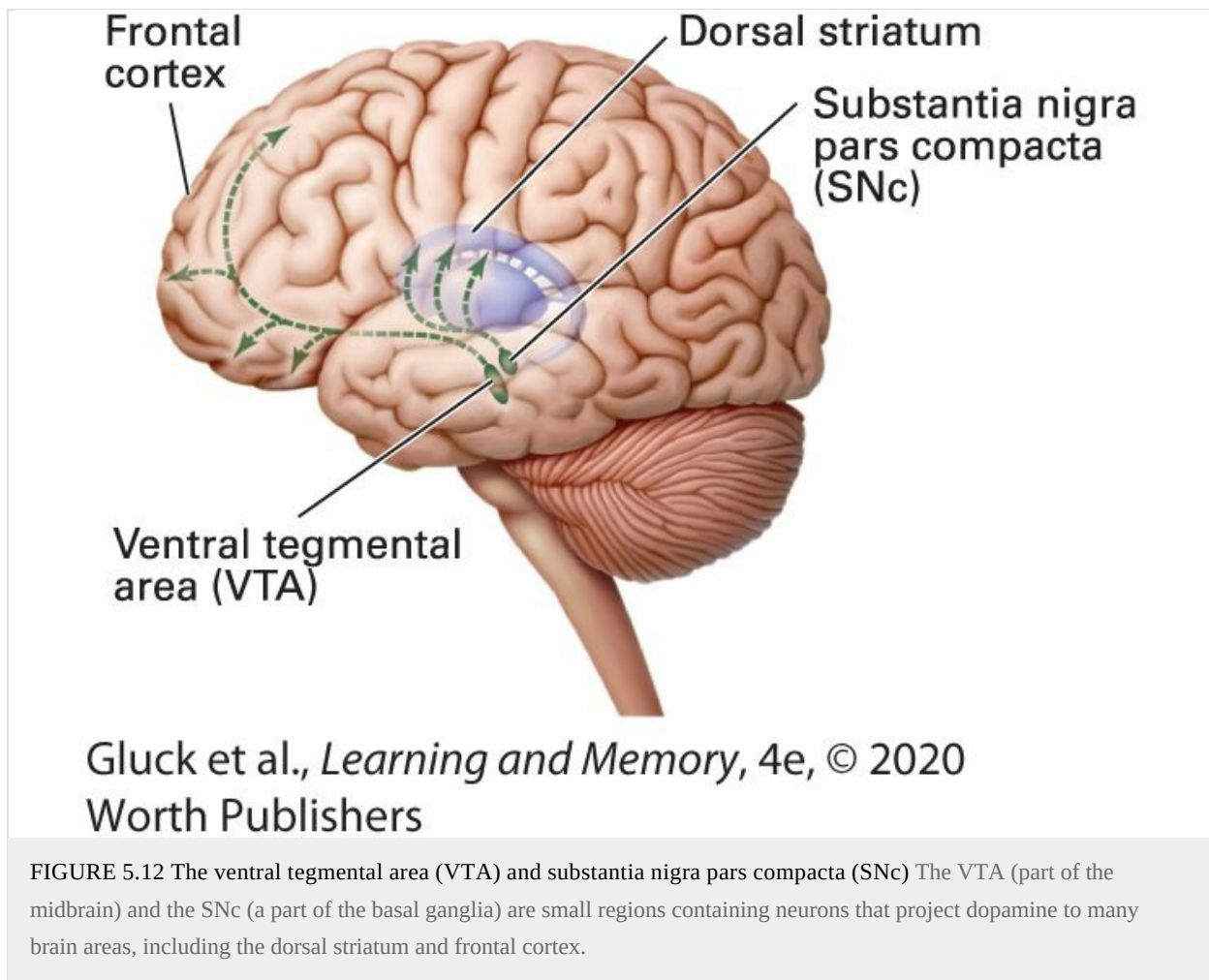
“Wanting” and “Liking” in the Brain

In 1954, James Olds was experimenting with delivering electrical stimulation to the rat brain. He inserted an electrode into an area that researchers now believe to have been the lateral

hypothalamus. Olds waited until the rat wandered into a certain corner of the experimental chamber, and then he applied a brief electrical current. A few minutes later, the rat came back to the same corner, and Olds provided a second stimulation. The rat caught on quickly and began to loiter in that corner of the chamber, apparently hoping for more electrical stimulation ([Olds, 1955](#)). Thus, electrical stimulation to this area of the brain seemed to be acting much like a reinforcer, increasing the probability of certain responses (in this case, hanging around the correct location).

Olds was intrigued, to say the least. He rigged a Skinner box so that the rats could press a lever to turn on the electrical stimulation. The rats were soon lever-pressing at a furious rate—as much as 700 presses per hour ([Olds, 1958](#)). If allowed, rats would press the lever continuously for up to 48 hours, until they collapsed from physical exhaustion! Given a choice between electrical stimulation and food, the rats would literally starve themselves, preferring the stimulation ([Routtenberg & Lindy, 1965](#)).

Later studies identified that rats would work for electrical stimulation in several brain areas, in particular the **[ventral tegmental area \(VTA\)](#)**, a small region in the midbrain of rats, humans, and other mammals ([Figure 5.12](#)). The electrodes in Olds's original studies were probably stimulating hypothalamic neurons with axons that project to the VTA, indirectly activating this area. Because VTA stimulation was such a powerful reinforcer, some researchers inferred that the rats “liked” the stimulation, and the VTA and other areas of the brain where electrical stimulation was effective became informally known as “pleasure centers.”



ventral tegmental area (VTA)

A brain region that contains dopamine-producing neurons projecting to the frontal cortex and other brain areas.

However, the idea of “pleasure centers” is an oversimplification. For one thing, rats pressing levers for electrical brain stimulation do not tend to act as if they are enjoying it; rather, they tend to become agitated and may bite the lever instead of simply pressing it, or they may show other behaviors, such as fighting, scratching the walls, or shredding the nesting material. This is more like the behavior of an excited animal than one who is enjoying food. Skinner, of course, would caution that we can’t infer what an animal might be feeling just by watching its behaviors. Nevertheless, some researchers suggested that electrical brain stimulation causes not pleasure but rather excitement or anticipation of reinforcement—much like the anticipation we experience when expecting a good meal or a big present ([Flynn, 1972](#)).

Currently, many researchers believe that we have separate brain systems for signaling **hedonic value**—meaning the subjective “goodness” of a reinforcer, or how much we “like” it—that are distinct from those signaling **motivational value**—meaning how much we “want” a reinforcer and how hard we are willing to work to obtain it. No matter how much we may “like” chocolate cake, most of us will not be very motivated to obtain more if we have just eaten three slices; similarly, Olds’s rats doubtless still “liked” food and rest, but they were more motivated to obtain electric brain stimulation, even when starving and exhausted. As these examples demonstrate, provision of a “liked” reinforcer is not enough to evoke responding. Only when “wanting” and “liking” signals are both present will the arrival of the reinforcer evoke responding and strengthen the $S^D \rightarrow R$ association.

hedonic value

In operant conditioning, the subjective “goodness” or value of a reinforcer.

motivational value

In operant conditioning, the degree to which an organism is willing to work to obtain access to a stimulus.

Dopamine: How the Brain Signals “Wanting”?

The neurotransmitter dopamine is produced by neurons in the ventral tegmental area (VTA), a part of the midbrain that projects to the frontal cortex (among other places); dopamine is also produced in the nearby **substantia nigra pars compacta (SNc)**, a part of the basal ganglia that projects to the striatum ([Figure 5.12](#)). As you read earlier in this chapter, the dorsal striatum is an important site of $S^D \rightarrow R$ association, and the orbitofrontal cortex is important for learning about predicted outcomes, so dopaminergic neurons in the VTA/SNc are a good place to start looking at how the brain signals motivational value.

substantia nigra pars compacta (SNc)

A part of the basal ganglia that contains dopamine-producing neurons projecting to the striatum.

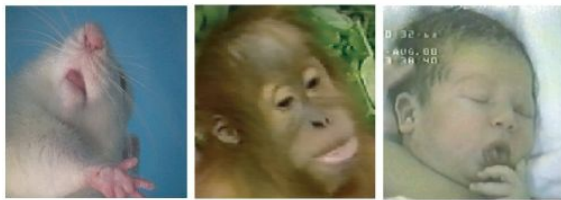
In rats, dopamine release from the VTA/SNc is triggered by encounters with food, sex, drugs of abuse, and secondary reinforcers. In humans, PET and fMRI studies have shown that presentation of juice, cocaine, money, humor, and even video games causes heightened activity in dopamine target sites such as the striatum ([Berridge & Robinson, 1998](#); [Knutson, Fong, Adams, Varner, & Hommer, 2001](#); [Mobbs, Greicius, Abdel-Azim, Menon, & Reiss, 2003](#)). Even in invertebrates, such as the sea slug *Aplysia*, dopamine is released in conjunction with positive reinforcement during operant conditioning ([Brembs, 2003](#); [Nargeot, Baxter, Patterson, & Byrne,](#)

[1999](#)).

Many researchers believe that dopamine does not simply signal hedonic value, or “liking.” For example, Parkinson’s disease is caused by the progressive death of dopamine-producing neurons, many of which project to the striatum. But when patients with Parkinson’s disease are asked to rate the perceived pleasantness of sweet and salty tastes, their ratings are the same as those of healthy people. Apparently, the dopamine reduction in these patients causes no loss of the ability to “like” pleasurable stimuli ([Travers et al., 1993](#)).

Similar results are obtained from nonhuman animals. Researchers can’t simply ask rats to rate the perceived pleasantness of different tastes. But just as a human child given a taste of sugar may lick her lips, a monkey or rat given sugared water will show rhythmic movements of the mouth and protrusion of the tongue. This is sometimes called the hedonic, or “yum,” reaction. On the other hand, a bitter taste produces a different cluster of responses: mouth gapes, nose wrinkling, shakes of the head, and wiping of the face with paws or hands—an aversive, or “ugh,” reaction that appears remarkably similar in humans, monkeys, and rats. Researchers can use the “yum” and “ugh” reactions to infer degree of liking in nonhuman animals (and in human infants). Rats given injections of a drug that destroys dopaminergic neurons exhibit hedonic (“yum”) and aversive (“ugh”) responses that are just as strong as or stronger than those of control rats ([Berridge & Robinson, 1998](#)). This suggests that rats with damaged dopamine systems continue to “like” food just as much as control rats do. What seems to change is their willingness to work for it.

Hedonic facial expressions (“yum”)



Aversive facial expressions (“ugh”)



Kent Berridge

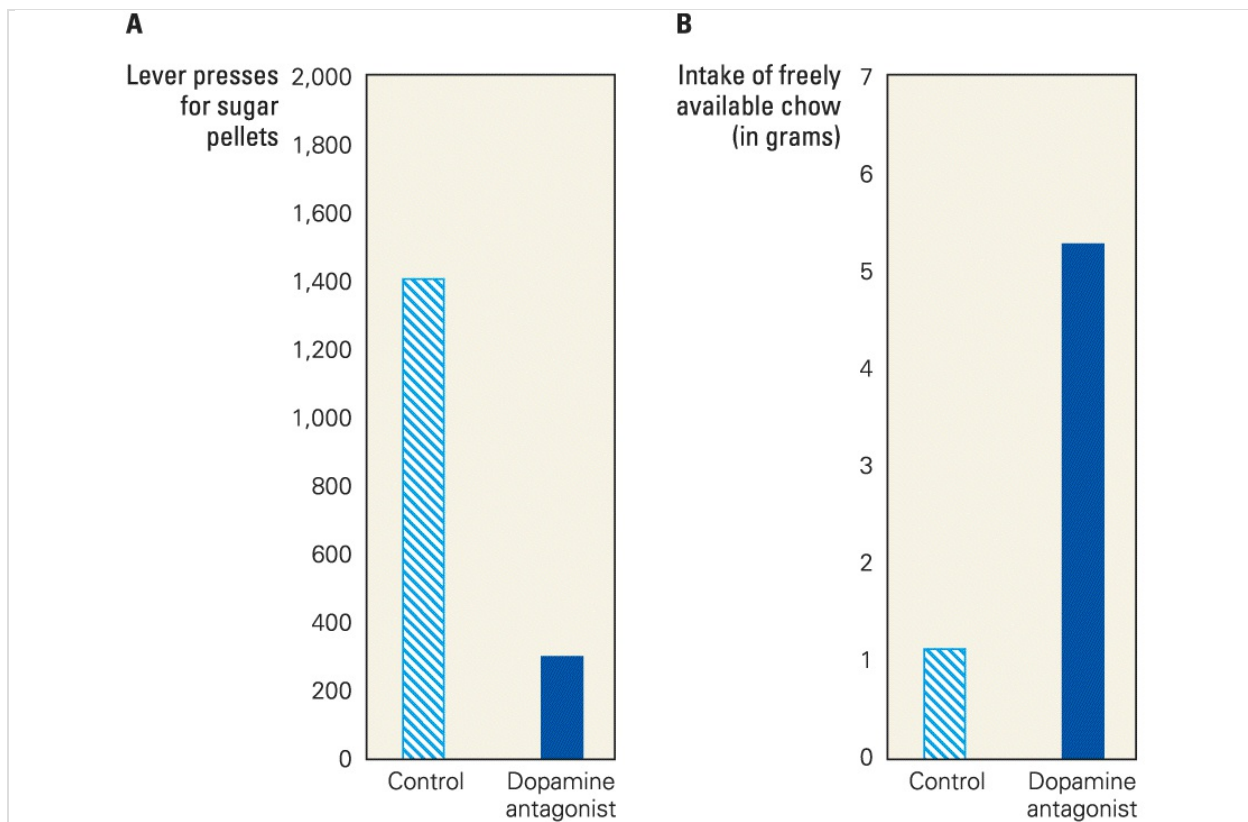
Given a pleasant, sweet taste, humans express characteristic hedonic, or “yum,” facial reactions that can include tongue protrusion (“licking one’s lips”); given an unpleasant, bitter taste, humans show aversive, or “ugh,” facial reactions that can include lip retraction (gaping) and wrinkling of the nose. Some of the same reactions can be observed in other species, such as orangutans and rats, allowing researchers to infer whether the animal likes or dislikes the taste. What purposes might tongue protrusion and gaping serve, besides providing a way to communicate our emotions to others?

The [incentive salience hypothesis](#) of dopamine function states that one role of dopamine in operant conditioning is to signal how much the animal “wants” a particular outcome—that is, how motivated the animal is to work for it. According to this hypothesis, the incentive salience of food and other reinforcers—that is, their ability to attract attention and motivate responding—is reduced in dopamine-depleted animals ([Berridge, 1996, 2007](#); [Berridge & Robinson, 1998](#)).

incentive salience hypothesis

The hypothesis that dopamine helps provide organisms with the motivation to work for reinforcement.

A good example of the incentive salience concept is demonstrated in behavioral economics experiments. For instance, most healthy rats prefer sugar pellets to rat chow. If they have to work for the pellets by lever-pressing but chow is freely available, they will typically choose to work for the pellets ([Salamone, Arizzi, Sandoval, Cervone, & Aberman, 2002](#)). Rats given a dopamine antagonist also prefer sugar pellets to rat chow, if both are freely available. But if those same rats have to work for the sugar pellets by lever-pressing, they work much less than control rats do ([Figure 5.13A](#)) and mostly settle for eating the free chow instead ([Figure 5.13B](#)). In other words, given a choice between competing alternatives, normal animals will tend to choose their preferred reinforcer, even at the cost of a little extra work. In contrast, dopamine-depleted animals are still perfectly willing to eat the preferred food if it is placed in front of them, but they are not willing to work hard to earn it.



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FIGURE 5.13 Dopamine and incentive salience Most rats prefer sugar pellets to rat chow. If the chow is freely available but sugar pellets have to be “earned” by pressing a lever, control rats (pale blue bars) will spend most of their time working for sugar pellets (A) and will eat relatively little free chow (B). In contrast, rats given a dopamine antagonist (dark blue bars) are less willing to work for the sugar pellets and instead settle for eating more of the freely available chow.

Information from [Salamone et al., 2002](#).

Description

The graph on the left has Y-axis plotting Lever presses for sugar pellets, with values from 0 to 2000 in increments of 200 and X-axis labeled Control and Dopamine antagonist. The control group pressed the lever an average of 1400 times for sugar pellets, and dopamine antagonist only pressed the lever about 300 times. The graph on the right has Y-axis plotting Intake of freely available chow (in grams), with values from 0 to 7 and X-axis labeled Control and Dopamine antagonist. The control group consumed about 1 gram of free chow and dopamine antagonist consumed 5 grams.

In an even more extreme case, mice that have been genetically engineered to be completely unable to produce dopamine will not seek food at all, and they generally starve to death by about 20 to 30 days of age—even if pellets are placed directly in front of them ([Palmiter, 2008](#)). However, if the food is placed in their mouths, these animals will chew and swallow and even exhibit “yum” responses, indicating that they still “like” food and can consume it; they just lack

the motivation to obtain it. These mice can be “rescued” by infecting cells in the striatum with a recombinant virus that allows the cells to produce and release dopamine; afterward, the mice eat enough normal chow to maintain body weight without further intervention.

Dopamine seems to affect incentive salience in humans, too. For example, in one study, humans addicted to cocaine were given a drug that increases brain dopamine levels; these participants reported an increased craving for cocaine but no increase in the self-reported “high” from cocaine ([Haney, Foltin, & Fischman, 1998](#)). Thus, stimulating the dopamine system increases “wanting” but not “liking” of cocaine.

The dopamine system can also be stimulated naturally by exposing the organism to a stimulus that has had its “temptation power” boosted by previous association with reinforcement ([Berridge, 2012](#)). For instance, the sight of chocolate can stimulate intense desire in chocolate lovers, even if they are not particularly hungry; similarly, a cigarette smoker who is sincerely trying to quit may experience an overwhelming craving if he enters a room where he can see and smell others smoking. For this reason, many smokers and drug addicts who wish to quit try to avoid environments where they are likely to encounter people using the addictive substance.

In addition to dopamine’s role in “wanting,” there is also considerable evidence that dopamine plays a key role in monitoring *reward prediction error*, meaning the degree to which an outcome is unexpected ([Schultz, 1998, 2015](#)). Reward prediction error information is critical when an organism is learning how to predict (and act to obtain) future reward. Remember the experiment, described in [Figure 5.10B](#), in which the monkey learned that picture 1 predicted upcoming grape juice, while picture 2 predicted upcoming orange juice? Imagine that now picture 1 is followed by orange juice (surprise!). When the unexpected outcome occurs, the monkey’s dopamine neurons spike sharply—in effect, signaling that something new and unexpected is happening. This signal travels to the orbitofrontal cortex (along with other brain areas), facilitating updates to $S^D \rightarrow R \rightarrow O$ associations to reflect the new reality, so that the monkey can continue to predict which responses will achieve its preferred outcomes.

Endogenous Opioids: How the Brain Signals “Liking”?

If dopamine signals “wanting,” then what signals “liking” in the brain? Probably the best-studied candidate is the opioid system. Opiate receptors in the brain were discovered quite by accident in

the 1970s by researchers trying to figure out how heroin and morphine work. Heroin and morphine belong to a class of drugs called *opiates*, which bind to a class of neuronal receptors called *opiate receptors*. Rather than assume that the brain evolved special receptors to respond to heroin and morphine, researchers suspected there might be naturally occurring brain chemicals that also activate the opiate receptors—and indeed there are. The [endogenous opioids](#) are naturally occurring neurotransmitter-like substances that have many of the same effects as opiate drugs. (The word *endogenous* means “originating on the inside”; *opiod* means “opiate-like.”) Endogenous opioids are distributed throughout the central nervous system, and when released into the body, they have a wide range of effects, such as reducing the normal perception of pain and producing feelings of euphoria.

endogenous opioid

Any of a group of naturally occurring neurotransmitter-like substances that have many of the same effects as heroine, morphine, and other opiate drugs; may help signal the hedonic value of reinforcers in the brain.

Although there is still a great deal to be learned about the endogenous opioids, many researchers believe these substances may mediate hedonic value, or “liking.” If so, the reason that heroin and morphine are so intensely pleasurable is that they happen to activate the same brain receptors as the endogenous opioids.

For example, morphine makes sweet food taste sweeter and bitter food taste less bitter ([Rideout & Parker, 1996](#)). It can also make pain feel less painful; morphine is used medically for patients who are enduring extreme, long-term pain (in cases where the benefits of relieving suffering outweigh the risks of morphine addiction). These patients usually report that they still feel the pain but that it does not trouble them as much as it did before.

Endogenous opioids are released in response to primary reinforcers, such as food, water, and sex, and they may be released in response to secondary reinforcers and pleasurable behaviors, too ([Le Merrer, Becker, Befort, & Kieffer, 2009](#)). Differences in the amount of endogenous opioid released and in the types of opiate receptors they activate may help determine an organism’s preference for one reinforcer over another ([Le Merrer et al., 2009](#)). This would contribute to effects such as the ones shown in [Figure 5.3](#), where infants will suck harder to obtain sweetened water even though plain water satisfies thirst just as effectively. Just like infants, rats normally prefer sweetened water to plain water, but rats given the opioid antagonist naloxone choose the sweetened water much less often than do control rats ([Hayward, Schaich-Borg, Pintar, & Low,](#)

[2006](#))—as if they no longer “like” the sweetness as much.

How Do “Wanting” and “Liking” Interact?

If dopamine signals “wanting” and the endogenous opioids signal “liking,” how might these two brain systems interact to influence behavior? The answer is not yet clear. One possibility is that endogenous opioids may modulate dopamine release. For example, some neurons in the VTA have opiate receptors on their dendrites that, when activated, could affect those neurons’ normal tendency to release dopamine. In this way, the endogenous opioids could signal “liking,” which in turn could affect the VTA’s ability to signal information about “wanting.” But other studies have suggested that different subpopulations of dopamine neurons might exist, conveying salience (“wanting”) and valence (“liking”) separately ([Matsumoto & Hikosaka, 2009](#)). The picture is complicated because some drugs, such as heroin, may manipulate both pathways—activating the “liking” system to produce a pleasurable high, while also activating the “wanting” system to produce a craving for more of the drug and the high.

Punishment Signaling in the Brain

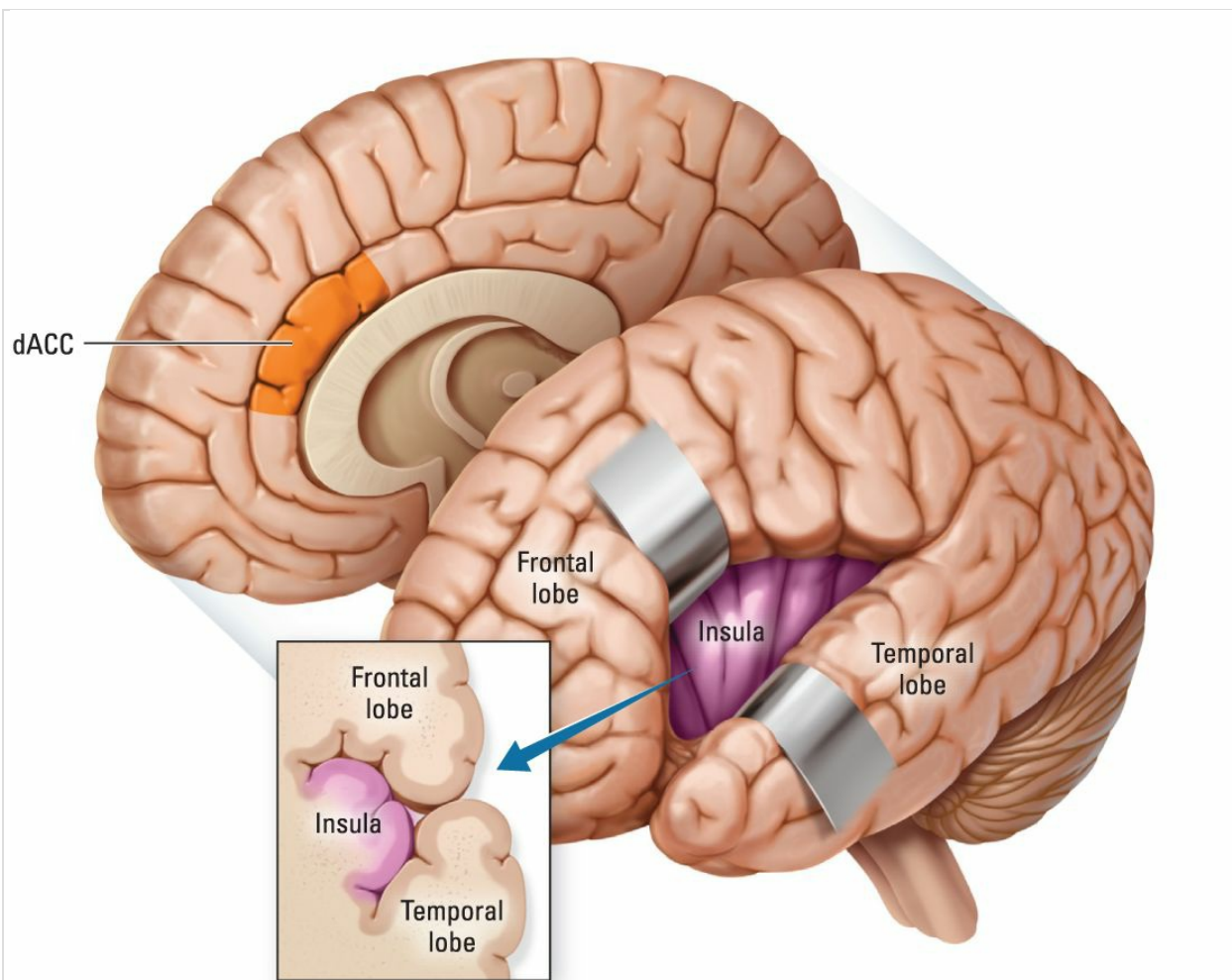
If the dopamine and opioid systems help code “liking” (hedonic value) and “wanting” (motivational value) of reinforcers, then what codes the aversive value of punishers? So far, there does not appear to be just one, unique “pain center” in the brain. Rather, both physical and emotional pain can activate multiple pathways and systems in the brain.

Physical pain often begins in the skin or musculature, where specific receptors called *nociceptors* respond to intense pressure, heat, or other stimulation that can cause damage. Messages from these receptors pass through the brainstem and thalamus to reach somatosensory areas in the cortex, such as the primary somatosensory cortex (S1; introduced in [Chapter 2](#) and illustrated in [Figure 2.8](#)). Brain-imaging studies have shown that the more intense the pain, the more activity in S1. When you shower, for example, as the water gets hotter, you will have more activity in S1. But although S1 encodes the physical location and intensity of pain, it does not encode how bad the pain “feels”—the affective component of pain. If you have spent all day freezing outside in the snow, standing under that same very hot shower may actually feel good rather than painful. Similarly, a man swallowing wasabi-flavored snacks may gasp for breath and wipe away tears—and then reach for another handful to do it again. Clearly, not all intense stimuli are aversive, and not all aversive stimuli cause physical pain: disgusting smells, loud discordant sounds, and social

rejection can all be highly aversive, even though no physical pain occurs.

The Insula: How Much Does It Hurt?

So how does the brain decide whether a particular stimulus is aversive? Several brain areas have been implicated, including the [insular cortex](#), or [insula](#), shown in [Figure 5.14](#). The insular cortex is located in the deep fold separating the temporal lobe from the parietal and frontal lobes, and it is important for our conscious awareness of our own bodies and emotional states. One subregion of the insular cortex, the *dorsal posterior insula*, plays a role in perception of physical pain, as well as in other negative emotional states, such as hunger, anger, and disgust ([Chang, Yarkoni, Khaw, & Sanfey, 2013](#); [Naqvi & Bechara, 2009](#)). For instance, functional neuroimaging has shown that the dorsal posterior insula is more active when research participants experience painful heat or cold ([Craig, 2003](#)), as well as when they experience social rejection—such as being excluded by other players in an online video game ([Eisenberger, Lieberman, & Williams, 2003](#)) or when viewing pictures of an ex-partner after an unwanted breakup ([Kross, Berman, Mischel, Smith, & Wager, 2011](#)). The degree of activation appears to be roughly proportional to the intensity of the punisher. So, for example, in a study where errors could be punished by loss of 5 cents or 50 cents, the insula showed more activity after a larger loss ([Hester, Murphy, Brown, & Skilleter, 2010](#)).



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FIGURE 5.14 The insular cortex (insula) and dorsal anterior cingulate cortex (dACC) The insula, which lies buried in the fold separating the temporal lobe from the parietal and frontal lobes, is implicated in conscious awareness and also plays a role in signaling the aversive value of stimuli. (The figure illustrates the surface of the frontal and temporal lobes being “pulled back” by metal clips to reveal the insula underneath in purple.) The dACC (shown in orange), which lies on the inner, or medial, surface of the prefrontal cortex, may play a role in the motivational value of punishers, helping select the actions we take in response.

insular cortex (insula)

A brain region that is involved in conscious awareness of bodily and emotional states and that may play a role in signaling the aversive value of stimuli.

Thus, just as the opioid system may signal pleasantness, or “liking,” the insula may help the brain determine unpleasantness, or “disliking.” In fact, when the insula is damaged, learning to avoid unpleasant outcomes is impaired. One study trained participants to play a simple computer game and found that people with damage to the insula could learn how to respond in order to

obtain reward (point gain), but they were impaired at learning to avoid punishment (point loss) compared with people with brain damage that spared the insula ([Palminteri et al., 2012](#)).

The Dorsal Anterior Cingulate Cortex: What to Do About It?

Once we have established that a stimulus is subjectively painful, the next step is to decide whether to do something about it. The [dorsal anterior cingulate cortex](#) (abbreviated [Figure 5.14](#)), has been implicated in the motivational value of pain—the degree to which pain can drive changes in behavior ([Craig, 2003](#)). For example, in one study, participants played a game in which they could win or lose money. During the game, neurons in the dACC responded both to errors that resulted in outright punishment and to errors that merely resulted in no reward—but there was more activity in the former case ([Simões-Franklin, Hester, Shpaner, Foxe, & Garavan, 2010](#)). Presumably, the worse the consequences of an error, the greater the motivation to change behavior.

dorsal anterior cingulate cortex (dACC)

A brain region that may play a role in the motivational value of pain.

The dACC also shows increased activation when study participants unexpectedly receive a reduced reward ([Bush et al., 2002](#); [Williams, Bush, Rauch, Cosgrove, & Eskandar, 2004](#)), and the activity level is predictive of whether participants actually change their response ([Williams et al., 2004](#)). Remember the phenomenon of negative contrast, in which monkeys and children refuse to work for a reward that is smaller than the one they expect? In effect, the smaller-than-expected reward is functioning as a punisher, leading to decreased responding. It is possible that the dACC is recognizing this negative contrast and signaling reduced motivation to work for the disappointing reward.

Thus, just as the brain has multiple systems for signaling the hedonic value and motivational value of reinforcers—“liking” via the opioid system and “wanting” via the dopamine system—the brain may also have multiple systems to signal the aversive value and motivational value of punishers, via brain areas such as the insula and dACC. However, much remains to be learned about how we process and respond to punishers in the brain.

[Interim Summary](#)

- The dorsal striatum is an important brain substrate for storing stimulus–response ($S^D \rightarrow R$) associations; these $S^D \rightarrow R$ associations may become relatively automatic and habitual.
- The orbitofrontal cortex may be an important brain substrate for storing response–outcome ($R \rightarrow O$) associations, including both specific identity of outcomes and expected time until those outcomes arrive.
- Reinforcers and punishers may activate neurons in the ventral tegmental area (VTA) and substantia nigra pars compacta (SNc), which project dopamine to the dorsal striatum, frontal cortex, and elsewhere. Interrupting these pathways, with lesions or drugs, disrupts operant conditioning.
- The incentive salience hypothesis suggests that dopamine modulates “wanting” rather than “liking,” determining how hard an organism is willing to work for a reinforcement. Dopamine may also signal “prediction error,” meaning the degree to which outcomes are unexpected or surprising.
- The endogenous opioids, which are mimicked by many highly addictive drugs, may signal the hedonic value (“liking”) of reinforcers.
- Parts of the insula may help us determine subjective “disliking” of painful physiological and psychological stimuli. The dorsal anterior cingulate cortex (dACC) may help determine the motivational value of punishers, which is used to guide changes in behavioral responding.

5.3 Clinical Perspectives

Through the brain's reinforcement system, animals are hardwired to seek and obtain the things they need for survival (food, water, sleep, etc.) and to avoid the things that threaten survival (pain, sickness, predators, etc.). Unfortunately, this powerful reinforcement system can go awry. As an example, consider the pleasure we feel when we eat fatty food, which ensures that we are sufficiently motivated to repeat the experience. The human brain evolved millennia ago, when our ancestors had to forage for food and could never be sure when they would find their next meal. Fat could be stored in the body and used for energy later, when food was scarce. Under these conditions, seeking out fatty foods was a good strategy for survival. In twenty-first-century America, however, food is easier for most of us to obtain, but our biological drives have not changed, and many—still motivated to obtain the taste of fatty foods—have become dangerously overweight.

Drug addiction is another way in which the reinforcement system can malfunction (or, rather, function only too well). [Chapter 4](#) explains how classical conditioning can contribute to drug addiction, but another large piece of the addiction puzzle is operant conditioning: learned responding to obtain a particular kind of reinforcement. Insights from operant conditioning studies may deepen our understanding of addiction and lead to more effective treatments.

Drug Addiction

We all know people who are “addicted” to their morning cup of coffee, their afternoon chocolate bar, or even their favorite television show. Such people may experience intense cravings and even withdrawal symptoms if forced to go without. For instance, someone who drinks strong coffee every morning may experience a pleasurable alertness after she indulges; she may experience crankiness, sleepiness, headaches, or difficulty paying attention if she goes without; and she may have trouble kicking the habit if she decides to give it up. However, unless the coffee is causing harmful medical consequences or is otherwise interfering with her normal life (for example, if she failed to pay the rent because she's spending all her income at Starbucks), this would not be considered a serious addiction.

In contrast, **pathological addiction** is a strong habit (or compulsion) that is maintained despite known harmful consequences ([Everitt & Robbins, 2016](#); [Grant, Potenza, Weinstein, & Gorelick,](#)

[2010](#)). For instance, a person will be diagnosed as pathologically addicted to cocaine if she is unable to quit, suffers debilitating withdrawal symptoms between highs, and is obsessed with obtaining her next hit of the drug, to the point where she starts neglecting other aspects of her life—such as her family and her job—because nothing else is as important to her as cocaine. Alcohol can drive pathological addiction in the same way: an alcoholic may be unable to give up drinking, even though it has cost him his job, his health, and his family. Similarly, a rat that starves rather than cease lever-pressing to obtain electrical brain stimulation could be considered pathologically addicted to the stimulation.

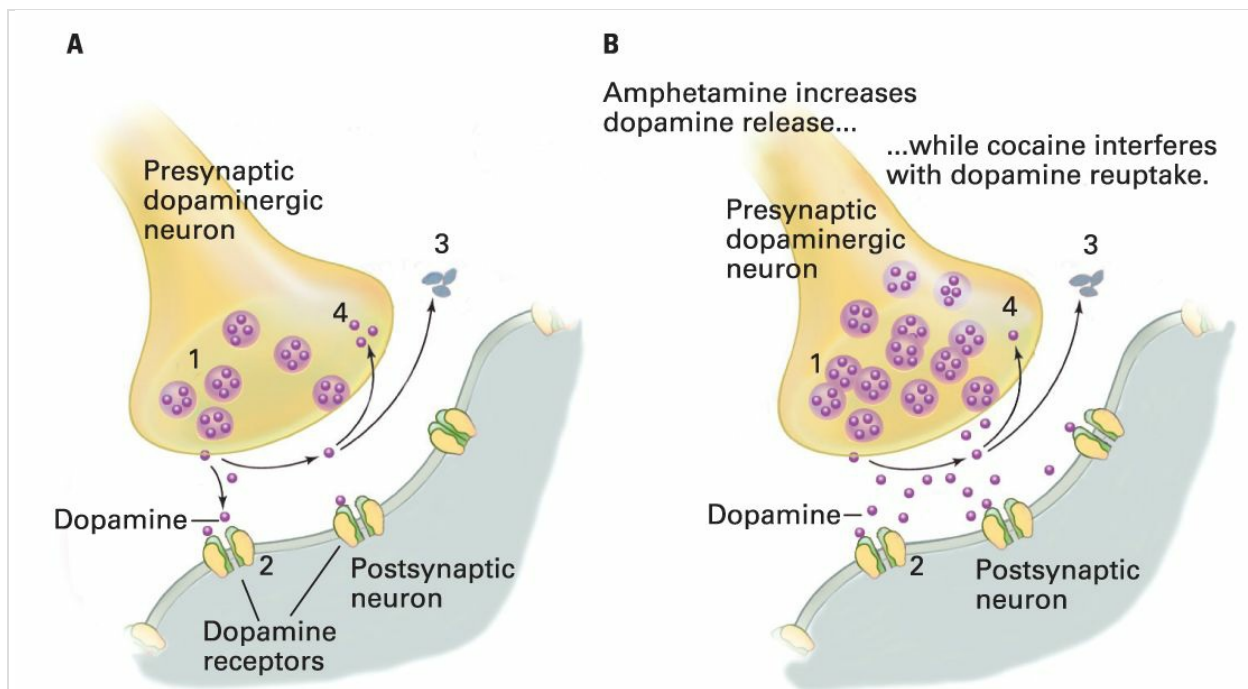
pathological addiction

A strong habit that is maintained despite harmful consequences.

Remember the college student Jamie who had to choose how to allocate his resources between going clubbing and purchasing expensive coffee ([Figure 5.7](#))? We can think of a person with addiction as having similar choices for allocating resources (time, money, effort) and increasingly choosing the addictive substance at the expense of all other options.

Many individuals with pathological addictions want to quit, and they try very hard to overcome their addictions. Unfortunately, there are several processes working against them. Addiction may involve not only seeking the “high” but also avoiding the adverse effects of withdrawal from the drug. In a sense, the high provides a positive reinforcement, and the avoidance of withdrawal symptoms provides a negative reinforcement—and both processes reinforce the drug-taking responses.

Many highly addictive drugs are opiates, meaning that they target opiate receptors in the brain. Heroin and morphine are two examples of opiate drugs. Other commonly abused drugs, including amphetamines and cocaine, work by increasing brain dopamine levels. As explained in [Chapter 2](#), neurons communicate when a presynaptic (or sending) neuron releases molecules of a neurotransmitter into the synapse, and these neurotransmitter molecules activate receptors on the postsynaptic (or receiving) neuron ([Figure 5.15A](#)). Amphetamine causes dopaminergic neurons to release higher levels of dopamine. Cocaine works by blocking dopamine reuptake, and dopamine remains in the synapse longer before being reabsorbed. In both cases, the effect is to increase the amount of dopamine available to activate the postsynaptic neuron ([Figure 5.15B](#)).



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FIGURE 5.15 The effects of amphetamine and cocaine on dopaminergic neurons (A) A presynaptic dopamine-producing neuron releases dopamine into the synapse (1). These molecules activate dopamine receptors on the postsynaptic neuron (2). Unused molecules are broken down (3) and taken back into the presynaptic neuron, a process called reuptake (4). (B) Amphetamine works by causing dopaminergic neurons to make and release more dopamine (1). Cocaine works by blocking the reuptake of unused dopamine molecules (4). Both drugs thus increase the amount of dopamine in the synapse, increasing the chance that dopamine molecules will activate receptors on the postsynaptic neuron (2).

Description

The first illustration A shows a labeled diagram of a presynaptic dopamine-producing neuron. The majority of Dopamine is released from presynaptic dopaminergic neuron into the synapse. It reaches Dopamine receptors and Postsynaptic neuron. A few dopamine molecules are released into the presynaptic neuron.

The second illustration B shows a labeled diagram of from presynaptic dopaminergic neuron, with the release of amphetamine. A text reads, amphetamine increases dopamine release, while cocaine interferes with dopamine reuptake. A large amount of dopamine is released into the postsynaptic neuron.

Both amphetamine and cocaine can be used as reinforcers. Thus, for example, rats and mice will learn to lever-press vigorously for injections of amphetamine or cocaine ([McBride, Murphy, & Ikemoto, 1999](#)). Unfortunately, as [Chapter 4](#) points out, long-term drug use can cause physiological changes in the synapse, such that ever larger doses of the drug are needed to get the same effect.

One interesting aspect of cocaine and amphetamine use is that, although “liking” seems to be critical in the early stages of drug use, people with long-term addictions often report that they no longer experience an appreciable high from the drug but crave it anyway—as if their “wanting” system has disconnected from their “liking” system and has run amok. In fact, individuals who have taken a dose of cocaine or amphetamine do not typically report feeling happy or pleased; rather, they feel aroused or excited. These facts fit nicely with the incentive salience hypothesis, which proposes that dopamine is involved in “wanting” but not necessarily in “liking.”

A number of studies have shown that the insula is among the brain areas activated when drug abusers experience cravings; this has been shown for cocaine, alcohol, opiates, and nicotine. In addition, several intriguing studies indicate that for cigarette smokers who happened to suffer stroke damage to the insula, their addiction to cigarettes was practically eliminated ([Gaznick, Tranel, McNutt, & Bechara, 2014](#); [Naqvi, Rudrauf, Damasio, & Bechara, 2007](#); [Suñer-Soler et al., 2012](#)). Similarly, animals that have been addicted to amphetamine and are then administered drugs to temporarily inactivate the insula show disruption of their drug-seeking behavior ([Contreras, Ceric, & Torrealba, 2007](#)). These studies and others like them suggest that the representation of negative feelings (cravings and withdrawal) by the insula helps maintain the addiction.

In summary, drug addiction is currently thought to result from several factors ([Robinson & Berridge, 2000](#)), including positive reinforcement (the pleasurable high), negative reinforcement (withdrawal avoidance), and malfunction of the dopaminergic “wanting” system (the craving). Of course, the relative role of each of these factors varies among individuals, as a function of genetics, personality, and experience. Some people become strongly addicted to drugs after a single use, some become addicted over time, and some can use drugs over a long period without developing pathological addictions ([Lüscher, 2016](#)). As yet, there is no way to predict whether a particular individual who starts using a particular drug will become addicted, and it is also impossible to know how difficult it will be for a particular individual to break free of addiction.

Behavioral Addiction

For some people, behaviors such as skydiving or winning at gambling can provide highs that are just as reinforcing as drugs—and just as addicting. **Behavioral addictions** are addictions to behaviors (as opposed to drugs) that produce reinforcements or highs, as well as cravings and

withdrawal symptoms when the behavior is prevented.

behavioral addiction

Pathological addiction to a behavior (rather than to a pharmacological substance).

Perhaps the most widely agreed-upon example of a behavioral addiction is compulsive gambling. Many people gamble occasionally, buying a weekly lottery ticket, playing online poker once in a while, or spending their vacations in Las Vegas or Atlantic City. These people pay their money, have their fun, and then walk away. But other people get hooked: they start to gamble more and more often, risking progressively larger sums of money, until nothing is as important as the chance to gamble. Up to about 1.6% of the general population may suffer from compulsive gambling at some point in their lifetime, and the problem may be even more widespread in particular groups, such as African Americans and college students ([Potenza, Kosten, & Rounsaville, 2001](#)).



Daniel Allan/The Image Bank/Getty Images

For some, gambling is an occasional, harmless pastime. For others, it can become a behavioral addiction. How would you decide whether a friend's frequent gambling had crossed the line to become a pathological addiction?

Skinner suggested that one reason gambling is so seductive is that it is reinforced on a VR schedule: you can never be sure when the next big payoff will come, which makes it easy to talk yourself into playing just one more time, then just one *more* time ... ([Skinner, 1953](#)). Each big win provides a powerful reinforcement of the gambling behavior.

Gambling is not the only behavior that can affect the brain's reinforcement system and become addictive. Other behavioral addictions include compulsive eating, sex addiction, compulsive exercising, kleptomania, compulsive shopping—the list goes on and on. In each of these cases, the addicted person experiences a high from the behavior, followed by craving for another high,

and withdrawal symptoms if the behavior is prevented.

A newly recognized form of behavioral addiction is *Internet gaming disorder* (IGD). Experts estimate that more than 90% of U.S. children and teenagers play video games, and somewhere between 1 and 9% of them may be addicted to gaming ([Gentile et al., 2017](#)). It is important to note that—as with other addictions—heavy game play alone does not necessarily mean that an individual is addicted. Rather, individuals with IGD are preoccupied with gaming to the extent that it has become the dominant activity in daily life; they lose interest in real-life relationships and other forms of entertainment, and they may exhibit withdrawal symptoms if access to gaming is taken away.

Behavioral addictions seem to entail dysfunction in the same brain substrates that are affected by drug addictions. Functional neuroimaging (fMRI) research has documented that gambling activates the brain in much the same pattern as seen when people with cocaine addiction receive an infusion of the drug ([Breiter, Aharon, Kahneman, Dale, & Shizgal, 2001](#)). Similarly, mice that have been selectively bred for overeating show greater concentrations of dopamine in the dorsal striatum, as do mice that have been selectively bred for excessive exercising ([Mathes et al., 2010](#)).

Other evidence for the similarity between drug addiction and behavioral addiction comes from patients with Parkinson's disease, who gradually lose dopamine-producing neurons. Patients with Parkinson's disease often benefit from treatment with dopamine agonists, drugs that “mimic” dopamine by activating dopamine receptors in the brain. Curiously, a small percentage of patients treated with high doses of dopamine agonists suddenly develop serious gambling problems ([Santangelo, Barone, Trojano, & Vitale, 2013](#)). Apparently, in these individuals, the dopamine agonists disrupt the brain's “wanting” system in much the same way as shown in [Figure 5.15](#) for amphetamine and cocaine, reinforcing the problem behavior. Often, the gambling problem can be cured simply by switching the patient to a different type of medication.

Together, these results suggest there is a general reinforcement system in the brain that is activated in similar ways for different categories of reinforcers, including primary reinforcers (food and water), secondary reinforcers (money), drugs (cocaine and amphetamine), and behavior (gambling and gaming) ([Breiter et al., 2001](#)). If so, a better understanding of the

biochemical and behavioral principles underlying drug addiction may also help in the treatment of individuals suffering from behavioral addictions.

Treatments for Addiction

The intensity of the cravings experienced in addiction, not to mention the often excruciating physical pain of withdrawal, make it impossible for many people to break free of addiction without outside help. Currently, in the United States, the vast majority of treatment plans for addiction involve cognitive therapy, which can include one-on-one sessions with a psychologist as well as self-help sessions with a support group (such as Alcoholics Anonymous and its many spin-off organizations—Gamblers Anonymous, Narcotics Anonymous, Overeaters Anonymous, and so on). Medical treatment may also help. For example, naltrexone is a drug that blocks opiate receptors, presumably decreasing the ability of heroin to bind to those receptors and cause a hedonic reaction. In some studies, people with heroin addiction who had undergone detoxification were able to stay off heroin longer if they received continuing treatment that included naltrexone ([Kirchmayer et al., 2002](#); [Rawson & Tennant, 1984](#)). Compulsive gamblers have also reported reductions in gambling urges following naltrexone treatment ([Grant, Kim, & Hartman, 2008](#)).

One way to interpret the effect of naltrexone is by considering addiction as a strong $S^D \rightarrow R \rightarrow O$ association in which sets of environmental stimuli (S^D) trigger the addictive behavior (R), resulting in the reinforcing outcome (O) of a “high” or a reduced craving or both. If the response R no longer produces the outcome O, then the frequency of R should decline. Naltrexone works by blocking the brain’s reinforcement system, so that subsequent heroin (or gambling) will no longer produce the outcome that it used to. As a result, the response (drug taking or gambling) should extinguish.

Unfortunately, treatment with naltrexone has drawbacks as well. One is that naltrexone must be taken daily. Another is that it does not reduce the person’s craving (“wanting”) for heroin. As a result, a recent review of treatments for people addicted to heroin or other opiate drugs concluded that oral naltrexone treatment was not statistically superior to placebo in preventing relapse ([Minozzi et al., 2011](#)).

The principles of operant conditioning have also suggested several other behavioral therapies to

help fight addiction. One conditioning-based technique for combating addictions and habits is *distancing*, or avoiding the stimuli that trigger the unwanted response. For example, a cigarette smoker who is struggling to quit and who gets the urge to light up whenever she hangs out with friends who are heavy smokers should try to avoid those situations. If the S^D is never present, the R may never be triggered.

Another technique is *differential reinforcement of alternative behaviors* (DRA), which you read about earlier in this chapter. If a smoker makes it through a whole week without a cigarette, she can reinforce her own abstinent behavior by treating herself to a favorite food or activity. Friends can help by praising the nonsmoking behavior; one aspect of Alcoholics Anonymous is the social reinforcement provided by the group for each week the alcoholic stays sober. Some programs for heroin addicts reinforce abstinence with actual monetary vouchers, providing yet another form of reinforcement for the alternative behavior of not using the drug ([Preston, Umbricht, & Epstein, 2000](#)).

A final conditioning-inspired technique is *delayed reinforcement*: whenever the smoker gets the urge to light up, she can impose a fixed delay (e.g., an hour) before giving in to it. Recall that increasing the delay between response and outcome weakens learning ([Figure 5.4](#)). Imposing long delays between cravings and cigarettes may similarly weaken the association; it will also, by default, reduce the total number of cigarettes smoked per day.

These and other approaches can be used in combination to increase the chances of success. But even with all these therapies, addicted cigarette smokers (and alcoholics and drug users and gamblers) can still have a very hard time kicking their habits. Currently, the most successful approaches appear to be those that combine cognitive therapy (including counseling and support groups) with behavioral therapy based on conditioning principles—along with medication for the most extreme cases ([Grant et al., 2010](#)).

Interim Summary

- Addictive drugs hijack the brain's reinforcement system and can produce psychological as well as physiological addiction.
- Opiates such as heroin and morphine mimic the endogenous opioids that are believed to form the brain's "liking" system, while drugs such as amphetamine and cocaine affect the

dopamine system, which is thought to signal “wanting” or craving.

- In addition, addiction is driven by the desire to avoid the aversive symptoms of withdrawal; these aversive symptoms may be signaled by the insula.
- Behavioral addictions may reflect the same brain processes as drug addictions.
- Among the treatments for people with addictions are cognitive therapies, medications, and behavioral therapies, including those based on principles learned from operant conditioning.

Synthesis

Recall the mine-sniffing rats you met in the introduction to this chapter. Their trainers teach them to scamper across a minefield to detect and signal buried explosives, in return for clicker reinforcement. It is a job the small animals can perform with little risk to themselves, while potentially saving thousands of humans from injury and death. The trainers use basic principles of operant conditioning, shaping one element of the behavior at a time and reinforcing desired responses with a clicker that acts as a secondary reinforcer. The principles of operant conditioning also apply to Becky, the little girl who was reinforced by an allowance for the behavior of keeping her room clean and who might be punished by time-outs at school for aggressive behavior during recess. Becky's parents will turn to the same principles again when it is time to housebreak a new puppy.

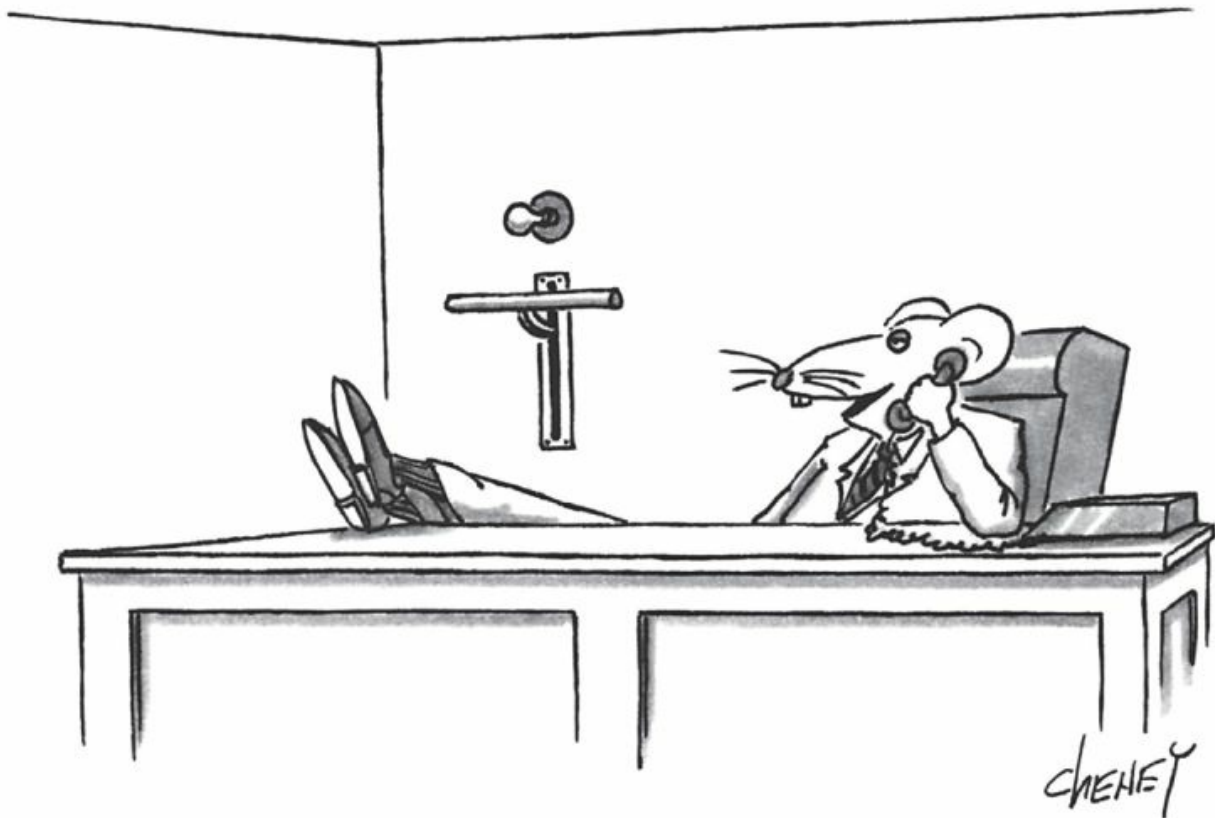
Operant conditioning is a powerful form of learning that can be applied to adult humans as well. You can even use it on yourself if you want to break a bad habit or reinforce good study practices. It is an approach that has been implicitly appreciated at least since the time of the ancient Greeks. In Aristophanes's play *Lysistrata*, first performed in 411 BC, the women of Athens agree to withhold sexual favors from their men until the men call off a frivolous war with Sparta—a clear example of negative punishment.

Many brain areas participate in operant conditioning. $S^D \rightarrow R$ associations may be stored in connections between the sensory cortex and the motor cortex and in the dorsal striatum; predictions about the outcome O that will follow R depend on frontal areas, including the orbitofrontal cortex. The brain also seems to have a general-purpose reinforcement system that includes the dopaminergic system, which may help an organism choose between competing responses with different predicted outcomes, and the endogenous opioids, which may signal hedonic value (“yum!”). Meanwhile, the insula helps signal the punishment value of stimuli (“ugh!”) and may provide this information to the striatum to help us learn which actions will result in avoiding or mitigating that punishment.

Many kinds of addiction, including drug addictions and behavioral addictions, result when chemicals or behaviors interfere with the brain's reinforcement system; many types of behavioral therapy for addiction apply operant conditioning procedures to help improve people's lives. Operant conditioning also forms a basis for understanding behavioral economics, the study of

how individuals allocate their time and energy among different available responses.

The bottom line: operant conditioning is not just for training rats in Skinner boxes. People use many operant conditioning techniques in daily life without even realizing it. By understanding the underlying principles, you can use them much more effectively.



*"Oh, not bad. The light comes on, I press the bar, they write me a check.
How about you?"*

The New Yorker Collection, 1993, Tom Cheney from cartoonbank. com. All Rights Reserved.

In what ways can human employment resemble operant conditioning? In what ways might it be different?

Chapter 5 Review

Know Your Key Terms

altruism

basal ganglia

behavioral addiction

behavioral economics

bliss point

chaining

concurrent reinforcement schedule

continuous reinforcement schedule

cumulative recorder

delay discounting

differential reinforcement of alternative behaviors (DRA)

discrete-trials paradigm

discriminative stimulus (S^D)

dorsal anterior cingulate cortex (dACC)

dorsal striatum

drive reduction theory

endogenous opioid

fixed-interval (FI) schedule

fixed-ratio (FR) schedule

free-operant paradigm

hedonic value

incentive salience hypothesis

insular cortex (insula)

matching law of choice behavior

motivational value

negative contrast

negative punishment

negative reinforcement

operant conditioning

orbitofrontal cortex

partial reinforcement schedule

[pathological addiction](#)
[positive punishment](#)
[positive reinforcement](#)
[post-reinforcement pause](#)
[primary reinforcer](#)
[punisher](#)
[punishment](#)
[reciprocal altruism](#)
[reinforcement](#)
[reinforcement schedule](#)
[reinforcer](#)
[secondary reinforcer](#)
[self-control](#)
[shaping](#)
[Skinner box](#)
[substantia nigra pars compacta \(SNc\)](#)
[token economy](#)
[variable-interval \(VI\) schedule](#)
[variable-ratio \(VR\) schedule](#)
[ventral tegmental area \(VTA\)](#)

Quiz Yourself

1. In operant conditioning, _____ signal whether a particular response will lead to a particular outcome. ([p. 177](#))
2. _____ refers to actions or behaviors that benefit another individual at the expense of some cost to the actor. Some such actions may reflect _____, or “tit-for-tat” behavior, in which one friend helps another with the expectation that the friend will reciprocate later on. ([pp. 202–203](#))
3. The _____ is a part of the brain that helps determine subjective values of punishers, such as whether the intense heat of a chili pepper on the tongue is perceived as pleasurable or painful. The _____ is a part of the brain that helps determine

the motivational value of punishment—that is, what we do about it. ([pp. 214–215](#))

4. _____ refers to a strong habit that is maintained despite harmful consequences; if the habit is a behavior, it is called a(n) _____. ([pp. 216, 218](#))
5. The part of the brain called the _____ contains dopamine-producing neurons that project to the _____, which is important for stimulus–response learning. A different area called the _____ contains dopamine-producing neurons that project to the frontal cortex and other brain areas. ([pp. 209–210](#))
6. The _____ theory states that learning is driven by an organism’s biological need to reduce innate drives to obtain primary reinforcers. ([p. 185](#))
7. In operant conditioning procedures, _____ is the process of providing outcomes for a behavior that increase the probability of that behavior occurring again in the future, whereas _____ is the process of providing outcomes that decrease the probability. ([p. 184](#))
8. _____ is the study of how organisms allocate their time and resources among possible options. ([p. 199](#))
9. In the operant conditioning technique known as _____, successive approximations to a desired response are reinforced. In the operant conditioning technique known as _____, organisms are gradually trained to execute complicated sequences of discrete responses. ([pp. 182–183](#))
10. _____ are stimuli such as food and sleep that can function as reinforcers due to their innate biological value to the organism; if these stimuli are paired with other stimuli that have no biological value, such as money or clicking sounds, those other stimuli can become _____. ([p. 185](#))
11. In a(n) _____ schedule, every instance of the response is followed by the consequence; in a(n) _____ schedule, only some responses are reinforced. ([p. 194](#))
12. An area of the prefrontal cortex called the _____ is important for learning to predict which outcomes follow particular responses. ([p. 206](#))

13. If a rat in a Skinner box expects that making a response will result in delivery of sugary water but the rat instead receives only plain water, the phenomenon of _____ predicts that the rat will respond less than if it had received plain water for its efforts all along. ([p. 185](#))
14. _____ are naturally occurring neurotransmitter-like substances that may help signal hedonic value (“liking”) in the brain. ([p. 212](#))
15. In a(n) _____ reinforcement schedule, an organism has a choice between multiple possible responses that may each lead to different outcomes. The _____ predicts that the organism will make each response at a rate proportional to how often that response is reinforced relative to the other choices. ([pp. 198–199](#))
16. In _____, organisms learn to make responses in order to obtain or avoid certain outcomes. ([pp. 175–176](#))
17. Training paradigms that can cause responses to become *less* frequent over time include _____, in which an undesirable element is delivered after a response, and _____, in which a desirable element is taken away after the response. ([pp. 191–192](#))
18. Training paradigms that can cause responses to become *more* frequent over time include _____, in which a desirable element is delivered after a response, and _____, in which an undesirable element is taken away after the response. ([p. 191](#))
19. In a fixed-ratio (FR) schedule of reinforcement, organisms typically give bursts of responding leading up to each reinforcement, followed by a(n) _____ before the next bout of responding begins. ([p. 194](#))
20. _____ refers to the subjective “goodness” of a stimulus. The amount of work an organism will be willing to do to obtain that stimulus depends on the _____ of that stimulus. ([p. 209](#))

Concept Check

1. A new kindergarten teacher wants to train her pupils to put away toys after playtime. Suggest at least three conditioning techniques she could use to train this behavior.
2. An employer wants to start testing her employees for drugs. She hopes that the threat of such tests will encourage employees to avoid drug use. According to the principles of conditioning, what would be the best way to schedule these drug tests?
3. Imagine that the police raid a house party and find the crowd taking an unfamiliar kind of drug. The users are sitting in front of a TV, enthusiastically munching on stale bread and laughing hysterically at an old sitcom. Even without submitting samples of the drug to a laboratory, what might we hypothesize about the mechanisms by which this drug works?
4. After the 9/11 terrorist attacks on the World Trade Center in New York City, sniffer dogs were used to search for survivors among the rubble. As the days went by and no survivors were found, handlers reported that the dogs showed signs of “depression,” or “not wanting to go to work in the morning.” To combat this, at the end of a long day of failure, handlers would arrange for the dogs to find a “survivor” (really, a healthy human hiding in the rubble) so that the dogs would end the day on a high note. Without invoking cognitive or emotional concepts, use the principles of operant conditioning to explain the utility of these “arranged finds.”

Answers to Test Your Knowledge

Is It Classical or Operant?

1. Whistling is the discriminative stimulus (S^D), birds arriving is the learned response (R), and birds eating the crumbs is the outcome (O). The birds do not get the crumbs (O) unless S^D is present and they make response R, so this is operant conditioning.
2. Thunder always follows lightning. This occurs whether or not Snoopy hides. Therefore, this is classical conditioning (CS = lightning, US = thunder, CR = hiding).
3. Presence of clouds is the discriminative stimulus (S^D), bringing the umbrella is the

learned response (R), and staying dry is the outcome (O). The outcome (O) does not occur unless S^D was present and Michael brought his umbrella (R), so this is operant conditioning.

4. Scalding water always follows flushing. This occurs whether or not Carlos flinches. Therefore, this is classical conditioning (CS = flush, US = hot water, CR = flinching).

Reinforcement Versus Punishment

1. (a) The learner is Lucy; (b) response R is a tantrum; (c) outcome O is obtaining candy; (d) S^D is seeing candy at the store because tantrums at other times will not cause Mother to purchase candy; (e) candy is added to Lucy's environment (addition = positive); (f) the frequency of Lucy's tantrum-throwing response increases—so this is a reinforcement paradigm. *Conclusion:* Lucy learns to throw tantrums to obtain candy. This is positive reinforcement.
2. (a) The learner is Susan; (b) response R is giving candy; (c) outcome O is stopping the tantrum; (d) S^D is the child's tantrum when she is refused candy because candy won't stop a tantrum unless a tantrum is already present; (e) the tantrum is taken away from Susan's environment (subtraction = negative); (f) the frequency of Susan's candy-giving response increases—so this is a reinforcement paradigm. *Conclusion:* Susan learns to give candy to stop the tantrums. This is negative reinforcement.
3. (a) The learner is Snoopy; (b) response R is crossing the boundary; (c) outcome O is the noise; (d) S^D is wearing the collar while in the yard because straying at other times will not produce the noise; (e) the noise is added to Snoopy's environment (addition = positive); (f) the frequency of Snoopy's straying response decreases—so this is a punishment paradigm. *Conclusion:* Snoopy learns to stop straying beyond the boundary. This is positive punishment.
4. (a) The learner is Miguel; (b) response R is drinking alcohol; (c) outcome O is revocation of playing privileges; (d) S^D is football season because Miguel will not be punished by the coach at other times of the year; (e) playing privileges are taken away from Miguel (subtraction = negative); (f) the frequency of Miguel's alcohol drinking behavior decreases—so this is a punishment paradigm. *Conclusion:* Miguel learns not

to drink alcohol during football season. This is negative punishment.

5. (a) The learner is Rachel; (b) response R is getting a stomachache (and telling the nurse about it); (c) outcome O is avoiding gym class; (d) S^D is school lunch on days when Rachel has gym class because sickness at other times does not affect gym class; (e) gym class is taken away from Rachel's environment (subtraction = negative); (f) the frequency of Rachel's sickness reporting behavior increases—so this is a reinforcement paradigm. *Conclusion*: Rachel learns to feel (or report) sickness in order to avoid gym class. This is negative reinforcement.

Reinforcement Schedules

1. FR; 5 homeworks = 1 toy. Note that the stars themselves are actually secondary reinforcers (on an FR 1 schedule, since 1 homework = 1 star).
2. VR; 20 phone calls = 2 sales on average, but the telemarketer can't be sure exactly when the next sale will come; it might come immediately after a previous call that also resulted in a sale.
3. VI; the couple can't be sure exactly when their table will be ready, so they check back periodically; only the last response after the table becomes ready will be reinforced. We would expect the couple to check infrequently in the first 20 minutes and more frequently as the expected 30-minute interval nears its end.
4. FI; money can be obtained for the first donation after a 2-week interval.
5. VI; the surfer can't be sure exactly when the next big wave will arrive, so the best policy is to hang around in anticipation, even immediately after a previous big wave.
6. FR; 1 gum = 1 negative reinforcement, which is removal of bad breath. This is also an example of continuous reinforcement, since each response R elicits the outcome O.
7. FI; the quiz occurs after a fixed 1-week interval, and good grades reinforce the studying behavior.
8. VR; 100 cards = 1 winner on average, but the very next card after a winner might also be a winner.



Chapter 6 Generalization, Discrimination Learning, and Concept Formation



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Behavioral Processes

Generalization: When Similar Stimuli Predict Similar Outcomes

Learning and Memory in Everyday Life: How Does Amazon.com Know What You Want

to Buy Next?

Discrimination Learning and Stimulus Control: When Similar Stimuli Predict Different Outcomes

Learning and Memory in Everyday Life: Sleep Better Through Stimulus Control

Beyond Similarity

Concept Formation, Category Learning, and Prototypes

Stereotypes, Discrimination, and Racism in Generalizations About Other People

Brain Substrates

Cortical Representations and Generalization

Generalization and the Hippocampal Region

Clinical Perspectives

Generalization Deficits in Schizophrenia

Altered Generalization in Autism Spectrum Disorder

SITTING IN HER HIGH CHAIR, 2-year-old Sabrina picks up a small piece of green broccoli from the plate of vegetables her father set before her and puts it in her mouth. Then, scrunching up her nose in disgust, she spits out the broccoli onto the floor and begins to pick through her plate for something else to try. She notices another piece that resembles the broccoli in shape and texture but that is white instead of green. If the white cauliflower tastes anything like the green broccoli, Sabrina would certainly want to avoid it. But she is hungry, and if the white cauliflower tastes good to her (in contrast to the green broccoli), she'd love to eat it. Sabrina faces what philosophers and psychologists have long understood to be a fundamental challenge for learning: how and when to generalize. **Generalization** is the transfer of past learning to new situations and problems.



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How might Sabrina's father persuade her to try different vegetables after seeing her reject the broccoli?

Description

A plate of broccoli and bell pepper salad is on the desk of the chair. Sabrina holds a fork in one hand and holds a green broccoli piece close to her mouth and nose with her other hand. Her eyes are wide open.

generalization

The transfer of past learning to novel events and problems.

If, after tasting (and spitting out) the green broccoli, Sabrina assumes that all the vegetables on her plate will taste terrible, she might find herself missing out on lots of wonderful foods to enjoy. On the other hand, if she assumes that only the vegetables that look exactly like the green broccoli are distasteful, she may suffer over and over again from eating related but not quite

identical vegetables whose flavors are similar. Sabrina is faced with the core issue of generalization: the need to find an appropriate balance between *specificity*, deciding how narrowly a rule applies (e.g., deciding that only the green, broccoli-like vegetables are nasty tasting), and *generality*, deciding how broadly a rule applies (e.g., assuming all the vegetables will taste nasty).

As her palate matures and her experience with different foods expands, Sabrina will learn to recognize the similarities and differences between vegetables—to treat some of them the same (generalization) while discriminating between those she considers delicious and those she finds unappetizing. **Discrimination learning** is the process by which animals or people learn to respond differently to different stimuli.

discrimination learning

The process by which animals or people learn to respond differently to different stimuli.

Generalization is seen in all the forms of learning and memory that are presented in this book. In rabbit eyeblink conditioning (a laboratory example of classical Pavlovian conditioning, described in [Chapter 4](#)), rabbits that are trained to blink to a 1,000-hertz (Hz) tone will generalize this learning, to some degree, to a 900-Hz tone and other tones of similar frequency. Likewise, when a pigeon has been trained through operant conditioning (described in [Chapter 5](#)) to peck a yellow light for food, it will generalize this learning, to some degree, from yellow to orange lights.

This chapter considers the behavioral and biological principles of generalization and discrimination and how they allow us to apply past learning to novel future situations. It also presents basic principles of **concept formation**, the process by which we learn about new categories of entities in the world, usually based on common features. Vegetable, for example, is a concept that helps us recognize a category of objects. Sabrina will eventually have a sense of what foods are vegetables and what foods are not. Our ability to function in a complex world and deal with novel challenges depends on our ability to form categories, recognize similarities between events or objects, make generalizations, and discriminate between members of different categories.

concept formation

The process by which we learn about new categories of entities in the world, usually based on common features.

Life never replays exactly the same events, with exactly the same objects or circumstances, that you have experienced before. To adapt—to survive and prosper—you need to apply what you have learned in the past to new situations that may be similar, but not identical, to what you have previously experienced.

6.1 Behavioral Processes

Two scenarios are possible, given Sabrina’s dilemma of whether or not to eat the white cauliflower that is so similar in shape and texture to the green broccoli she dislikes. Either eating the two vegetables will have the same outcome—and the cauliflower will taste bad to her—or eating the two vegetables will lead to different outcomes, and she’ll find the white cauliflower, unlike the green broccoli, to be yummy. These are special cases of the more general problem of generalization, schematized in [Table 6.1](#). The discussion in the next few pages is organized around this table, addressing in turn each of the four situations it describes.

TABLE 6.1 Alternatives for generalization about and discrimination between two stimuli

	Same outcome	Different outcomes
Similar stimuli	Similar stimuli → same outcome	Similar stimuli → different outcomes
	<i>Broccoli and cauliflower → nasty</i>	<i>Broccoli → nasty</i>
		<i>Cauliflower → yummy</i>
Dissimilar stimuli	Dissimilar stimuli → same outcome	Dissimilar stimuli → different outcomes
	<i>Broccoli and red peppers → nasty</i>	<i>Broccoli → nasty</i>
		<i>Red pepper → yummy</i>

As we examine each of these facets of generalization, we will see how psychologists’ efforts to understand them led to models that accomplished two things. First, the models allowed psychologists to formulate general principles of behavior and to predict, often with quantitative detail, the behaviors to be expected in a wide range of learning and memory experiments. Second, the models—originally posited as abstract theories about networks of associations—identified the basic brain mechanisms used to accomplish these processes (which are described in [Section 6.2](#)), and they did so years before science was able to verify their existence.

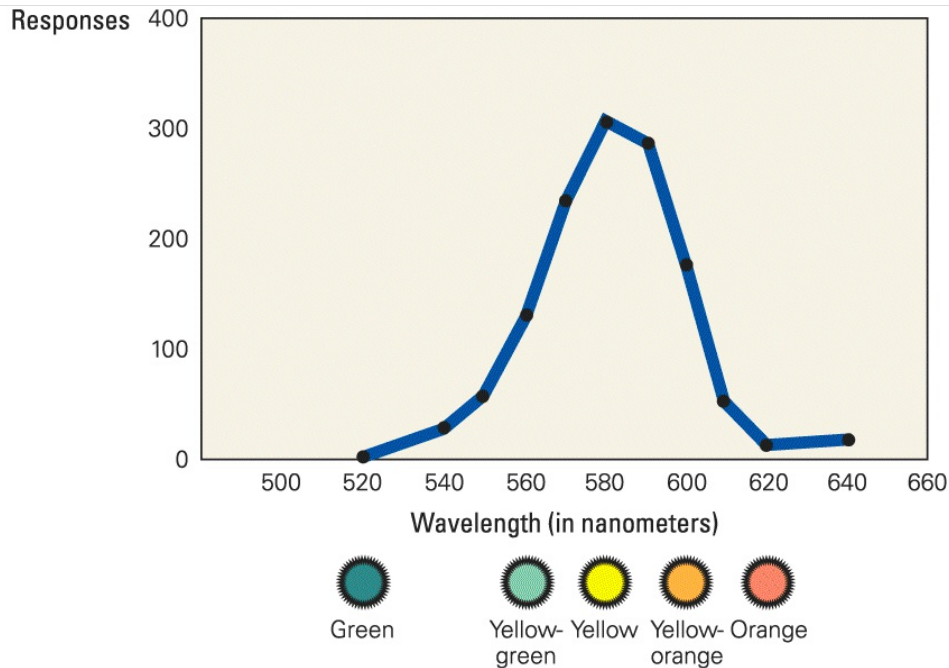
Our first discussion concerns the learning that occurs when similar stimuli predict similar outcomes or consequences, such as when a child decides that both the broccoli and the cauliflower taste nasty, corresponding to the top-left quadrant of [Table 6.1](#). The key questions that psychologists have asked about these situations are (1) when we know two things are

similar, what are our expectations for how likely they are to lead to the same outcome, and (2) can we construct a theoretical model that shows how people mentally represent the similarity of two entities, such as broccoli and cauliflower?

Generalization: When Similar Stimuli Predict Similar Outcomes

Using a situation comparable in structure to the problem of Sabrina and her vegetables, Harry Guttman and Norman Kalish trained pigeons to peck at a yellow light for food reinforcement ([Guttman & Kalish, 1956](#)). They then tested what the pigeons had learned by showing them, in each of a succession of test trials, a single colored light that was either green, yellow, orange, or one of the in-between colors yellow-green or yellow-orange. By counting how often the pigeons pecked at each color, the investigators were able to measure how the similarity of the color to the yellow training stimulus affected the amount of pecking. Because the sensation of color results from physically different wavelengths of light—for example, yellow light has a wavelength of 580 nanometers (nm)—researchers can compare colors in terms of wavelengths. In this case, the colors ranged along a physical continuum from green (520 nm) to orange (640 nm).

[Figure 6.1](#) shows how the pigeons responded. Not surprisingly, the pigeons pecked most at the stimulus on which they were trained: the yellow light, with its wavelength of 580 nm. However, the pigeons also responded to lights of other colors. Lights most similar to yellow (as measured by wavelength) produced the next-highest levels of responding. As the colors grew increasingly different from the original training stimulus (yellow), responding decreased rapidly.



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FIGURE 6.1 Stimulus-generalization gradients in pigeons Pigeons were trained to peck at a yellow light, having a wavelength of about 580 nm. When the pigeons were tested with other colors of light, their response rates decreased as the colors (wavelengths) departed further from the trained color.

Data from [Guttman & Kalish, 1956, pp. 79–88](#).

Description

The vertical axis is labeled responses, ranging from 0 to 400, in increments of 100. The horizontal axis is labeled wavelength (in nanometers), ranging from 500 to 660, in increments of 20. The wavelength (in nanometers) of green light is 520, yellow-green light is 560, yellow light is 580, yellow-orange light is 600, and orange light is 620. A line increases from (0, 520) and passes through (540, 20), (550, 50), (560, 120), and (570, 220) to reach a maximum at (580, 300). The line then decreases through (590, 290), (600, 180), (610, 50), and (620, 10), before ending at (640, 15). All values are estimated.

The graph in [Figure 6.1](#) is a **generalization gradient**, a curve showing how changes in the physical properties of stimuli (plotted on the horizontal axis) correspond to changes in responding (plotted on the vertical axis). The curve is called a “gradient” because it generally shows that an animal’s response changes in a graded fashion that depends on the degree of similarity between a test stimulus and the original training stimulus. After training in which a single stimulus (such as a light of a particular color) has been reinforced repeatedly, generalization gradients show a peak, or point of maximal responding, that corresponds to the original stimulus on which the animal was trained. This responding drops off rapidly as the test

stimuli become less and less similar to the training stimulus.

generalization gradient

A curve showing how physical changes in stimuli (plotted on the horizontal axis) correspond to changes in behavioral responses (plotted on the vertical axis).

From looking at a generalization gradient, you can deduce to what degree animals (including people) expect similar outcomes for stimuli that vary in some physical property, such as light wavelength. Thus, a generalization gradient is often taken to be a measure of the animal's or person's perception of similarity, in that if two stimuli are perceived as being highly similar (or identical), there will be significant generalization between them.

Generalization gradients have been studied for stimuli that vary in height, angle, size, or tone frequency. The generalization gradient in [Figure 6.1](#) represents typical behavior for many different stimuli and across a broad range of generalization experiments. The fundamental characteristic of these generalization gradients is that they decline rapidly with distance from the peak. Roger Shepard, an influential American cognitive psychologist, described this feature of generalization gradients as one of the most constant basic laws of psychology ([Shepard, 1987](#)).

Generalization as a Search for Similar Consequences

Why should a pigeon that has been trained to peck at a yellow light respond to an orange light at all? Could the pigeon be making a mistake—confusing the orange light and the yellow light or failing to tell them apart? Probably not. Pigeons can easily be trained to discriminate yellow from orange. One explanation for generalized responding is that pigeons (and all other animals, including humans) are savvy estimators of the probability of future events.

Imagine you are prospecting for gold, and you find a mountain stream that yields bountiful quantities of ore. After taking all the gold from that one stream, you wonder where you might find more. If you are optimistic, you imagine that you have stumbled onto a fabulous mother lode and that all the streams in the entire valley are filled with gold. At your most pessimistic, you realize the gold may be in only this particular stream. Perhaps the truth lies between these two extremes, and there is gold in just a few more streams in the valley.



Comstock/Getty Images

How might a prospector determine if the nugget he found is just a random single piece of gold or the beginning of a mother lode with lots more gold to be found?

Your initial discovery of gold in one stream does not tell you the size or shape of the gold-producing region or the location of its boundaries. All you know is that this one particular stream that yielded gold lies inside a gold-producing region of unknown extent. According to Roger Shepard, the fundamental challenge of generalization—for the gold miner, for 2-year-old Sabrina, and for Guttman and Kalish's light-pecking pigeons—is to identify the set of all stimuli that have the same consequence as the training stimulus. Shepard called this set the

[consequential region](#) (Shepard, 1987).

consequential region

A set of stimuli in the world that share the same consequence as a stimulus whose consequence is already known.

In Shepard's view, the generalization gradient in [Figure 6.1](#) reflects the pigeon's best estimate of the probability that novel stimuli will have the same consequence as a training stimulus. The pigeon, Shepard argues, is therefore not *confusing* a yellow-orange 600-nm light with the original yellow 580-nm light; rather, the pigeon, by responding at about 50% of its original rate to the yellow-orange light, is implicitly showing that it *expects*, based on what it learned from pecking the yellow light (which always resulted in food), that there is an even chance that pecking the yellow-orange light will yield the same food delivery. The shape of generalization gradients, Shepard argued, suggests that animals (including people) consistently expect that the chance that two stimuli will have the same consequence drops off sharply as the stimuli become more distinct. This implies that you can view generalization gradients as an attempt to predict, based on past experience, the likelihood that the consequences of one stimulus will also be the same as that of other, similar stimuli.

The Challenge of Incorporating Similarity into Learning Models

[Chapter 4](#) describes formal models of classical conditioning, such as the Rescorla–Wagner model, in which networks of associations (each with an associative weight) link stimuli (lights and tones) to outcomes (airpuffs or shocks). In the studies that led to these models, the stimuli were highly distinct, with little perceptual similarity between them. When two stimuli were employed, they were usually from two different modalities, such as a light and a tone. But what if the stimuli in a conditioning experiment are two tones of similar frequency or two lights of similar wavelength? The application of Rescorla and Wagner's associative learning model from [Chapter 4](#) to Guttman and Kalish's pigeon experiment (which used similarly colored lights) provides a good illustration of how easily the Rescorla–Wagner model falls apart and makes erroneous predictions unless you incorporate an additional assumption about how to represent the physical similarity of different stimuli.

When learning involves multiple cues in compound cue training, the Rescorla–Wagner model can be visualized as a simple one-layer network with links from the various cues to the possible

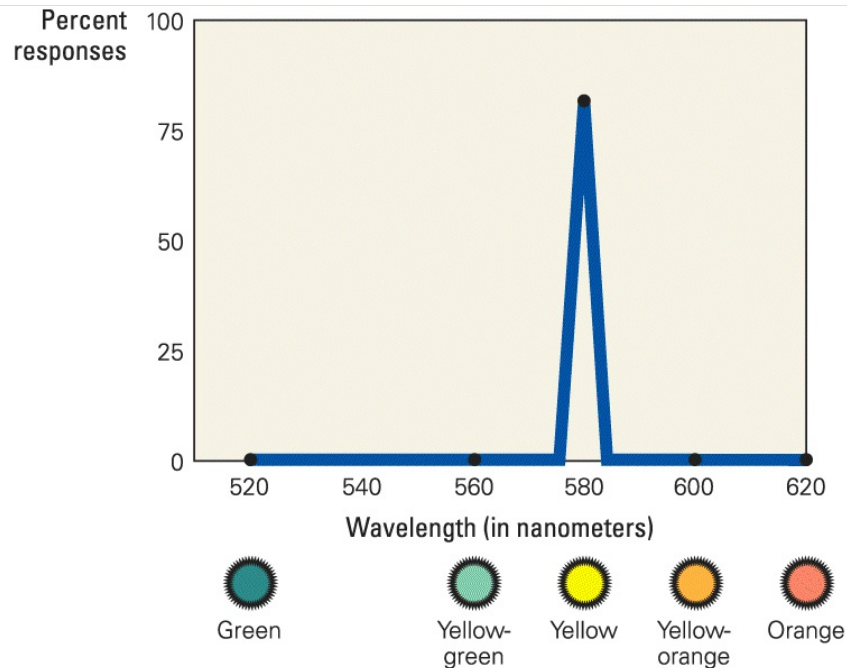
outcomes, as described in [Chapter 4](#) and illustrated in [Figure 4.11](#) ([Gluck & Bower, 1988](#)). Each trial is modeled by activating the corresponding input node for each stimulus cue (such as a tone), which then causes activation to travel through links (modulated by the associative weights) to an output node (e.g., the prediction of the airpuff). When activation from a given input node reaches an output node, it is added to the incoming activations of all the other active stimulus cues on that trial. Learning results when the associative weights on the links are modified at the end of each trial so as to reduce the likelihood of a future mismatch (or “error”) between the network’s prediction for an outcome (the activation in the output nodes) and the actual outcome that is presented as feedback on that trial by the experimenter.

Thus, as described in [Chapter 4](#), the Rescorla–Wagner model details a process whereby an animal or a person learns to minimize the difference between what actually happens (the airpuff) and the animal’s or person’s expectation of that outcome. It is the expectation of the airpuff outcome that generates an anticipatory conditioned response, such as an eyeblink. Therefore, in the [Chapter 4](#) applications of this model to classical conditioning, a tone and a light are identified as two distinct stimuli, each with an associative weight communicated from a corresponding input node to an output node that produces the conditioned response. To apply the same idea to Guttman and Kalish’s operant pigeon paradigm, we need five input nodes, one for each of the five discrete colors that might be presented.

[Figure 6.2](#) shows an attempt to test stimulus generalization (i.e., response to a stimulus because of its similarity to another stimulus) using a simple network model that has a single input node for each of five possible colors of light. This layer of nodes is an example of **stimulus representation**, the form in which stimuli are encoded in a theory or model and possibly in the brain itself. [Figure 6.2A](#) shows a particular type of stimulus representation called a **discrete-component representation**, meaning that each possible stimulus is represented by its own unique node in the model. The network also contains a single output node for the response and has weights that link the input nodes to the output node and that are modifiable by learning according to a learning algorithm such as the Rescorla–Wagner rule. Each input node in [Figure 6.2A](#) represents a different color of light (e.g., green, yellow, orange). Depending on the pattern of inputs, activity may be evoked in the output node; strong activity in this output node will cause the model to generate a response.

meaning there are no prior associations between any of the lights and the response. From then on, each training trial is modeled by activating the yellow-light input node, thereby triggering the output node or not, according to the associative weight from the yellow-light input node. This associative weight is then updated based on the difference between the actual output node activation and the desired, or ideal, output node activation (represented in [Figure 4.11](#) by the “teaching” node). In these trials, the ideal output node activation is 1 because a reward will follow every time the network generates a response to the yellow light cue. If the model is given repeated training trials of this sort, in which the yellow light is presented followed by a reward, then the weight of the association connecting the yellow input node to the output node will increase until it has a value close to 1.0. [Figure 6.2A](#) displays the weight from the yellow node as being thicker than the others to represent this greater weight.

[Figure 6.2B](#) illustrates what happens when this model is now tested with presentation of a novel yellow-orange light. There is still a strong 1.0 weight from the yellow node to the output node (because of the previous training), but that weight is not active (because a pure yellow light is not presented during this new test trial). Instead, only the yellow-orange input node is active, and it causes no activity in the output node because the weight from this input node is still 0.0. Thus, the model will produce no response to the yellow-orange light, even though that stimulus is very similar to the trained yellow light. In fact, this model will produce no response to any stimulus other than the original yellow training stimulus and therefore shows no generalization. The result would be a generalization gradient, as shown in [Figure 6.3](#): the model produces strong (about 80%) responding to the yellow test stimulus but no (0%) responding to any other color. This does not look like the more gently sloping generalization gradient obtained from Guttman and Kalish’s pigeons, seen in [Figure 6.1](#). Clearly, the model in [Figure 6.2](#) is wrong: it predicts a generalization gradient that is totally at odds with what Guttman and Kalish actually found.



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FIGURE 6.3 The generalization gradient produced by the discrete-component network of [Figure 6.2](#). The discrete-component network gives no response to the yellow-orange light, despite the similarity in color to the previously trained yellow light. In other words, this simple network fails to show a smooth generalization gradient like that shown by the pigeons in [Figure 6.1](#).

Description

The vertical axis is labeled percent responses, ranging from 0 to 100, in increments of 25. The horizontal axis is labeled wavelength (in nanometers), ranging from 520 to 620, in increments of 20. The wavelength (in nanometers) of green light is 520, yellow-green light is 560, yellow light is 580, yellow-orange light is 600, and orange light is 620. A line starts at (0, 520), passes through (560, 0), and remains constant until (575, 0). It then increases to reach a maximum at (580, 80). The line then decreases to (585, 0), passes through (600, 0), before ending at (620, 0). All values are estimated.

This analysis suggests that the simple models that use one discrete node for each different stimulus feature are limited in scope. Such discrete models are useful for describing, understanding, and predicting how organisms learn about highly dissimilar stimuli, such as a tone and a light, but they do not work as well with stimuli that have some inherent similarity, such as lights of different colors. Do we throw out the old models when we run into a problem like this and simply assume that the Rescorla–Wagner rule is wrong? Not necessarily. Instead, given this learning model’s otherwise wide range of success, researchers have tried to extend it

to account for generalization gradients. In this way, simple models can be considered to be preliminary steps toward a more complete understanding of learning and memory that builds, cumulatively, on past progress.

The learning model shown in [Figure 6.2](#) uses the simplest possible scheme for representing stimuli: a discrete-component representation in which each distinct stimulus is represented by a single input node that is then connected by a modifiable weight to an output node. Discrete-component representations are applicable to situations in which the similarity between cues is small enough that there is negligible transfer of response from one to another. This was the case in the experiments discussed in [Chapters 4](#) and [5](#), many of which used just a single tone or light. However, discrete-component representations fail in cases where stimuli have a high degree of physical similarity, since the models then produce unrealistic generalization gradients like the one shown in [Figure 6.3](#).

Before moving on, let's consider how different kinds of representations can have different degrees of usefulness, depending on the task—whether in our models, in our mental activities, or in the actual physical workings of our brain. Let's compare some of the different kinds of representations you use in everyday life. For instance, the letters in the words on this page represent sounds, and the words themselves represent ideas and concepts. To represent Rachel, a Rutgers University student, the government uses her nine-digit Social Security number, 015 88 6999; the university uses her student ID number; and the phone company uses her mobile phone number. Each of these representations was chosen to suit a particular need. However, what is appropriate for use as a representation in one context may not be appropriate in others. Of particular importance to our discussions in this chapter is the fact that different representations in different contexts yield different patterns of similarity: Rachel is most similar to Reyna when their names are spoken aloud (even if they have very different Social Security numbers). However, to the government, Rachel may be more similar to Elizabeth, whose social security number, 016 88 6999, differs by only one digit from Rachel's.

With this deeper appreciation for the effect of context on similarity between representations, we next explore how psychological theorists have grappled with the limitations of various stimulus representation schemes in learning models and how their solutions changed modern approaches to understanding the interplay between similarity, representation, and generalization. Meanwhile,

the “[Learning and Memory in Everyday Life](#)” box describes how companies such as [Amazon.com](#) have learned to use similarity to generalize for much the same adaptive purpose.

LEARNING AND MEMORY IN EVERYDAY LIFE

How Does [Amazon.com](#) Know What You Want to Buy Next?

Have you ever had the pleasure of meeting someone and discovering you have many interests and tastes in common? You both like the same obscure music, enjoy the same French films, and have read many of the same books. Finding someone like this is like finding your doppelganger (your phantom twin or double), and once you have found such a person and confirmed that you like the same books he mentioned, you would take very seriously his recommendation of a new book that is unfamiliar to you.

[Amazon.com](#) and other websites attempt to achieve much the same thing when they use a form of generalization to predict what books you might want to buy from them. They do it by trying to find as many of your doppelgangers as possible among their other customers. The process they use, called *collaborative filtering*, is an automatic filtering (or making of predictions) based on information from a large number of other people’s past behaviors. In essence, collaborative filtering works by creating a very detailed stereotype of what kind of reader you are.

Once [Amazon.com](#) has found a sufficiently large number of like-minded individuals among its customers, it collates their purchasing histories to find books some of them have not yet purchased but that were purchased by one or more other individuals with similar tastes. Thus, by profiling you and creating a mini-category of individuals similar to you, [Amazon.com](#) seeks to generalize from your past behaviors to predict what you might do in the future. In this way, [Amazon.com](#) and other websites not only increase their ability to sell large numbers of books but also help you discover the hidden gems you might miss if you never met your own personal doppelganger.

Shared Elements and Distributed Representations

Edward Thorndike’s *law of effect* (examined in [Chapters 1](#) and [5](#)) states that the probability of a response will increase or decrease depending on the outcomes that follow. Thorndike was well aware back in the 1920s of the limitations of discrete-component representations of stimulus features. He proposed that stimulus generalization occurs because of the elements *shared* by similar stimuli ([Thorndike, 1923](#)). For example, in Thorndike’s view, yellow and yellow-orange are not two totally separate stimuli. Rather, he argued, they are each composed of many distinct elements, some shared and others not shared.

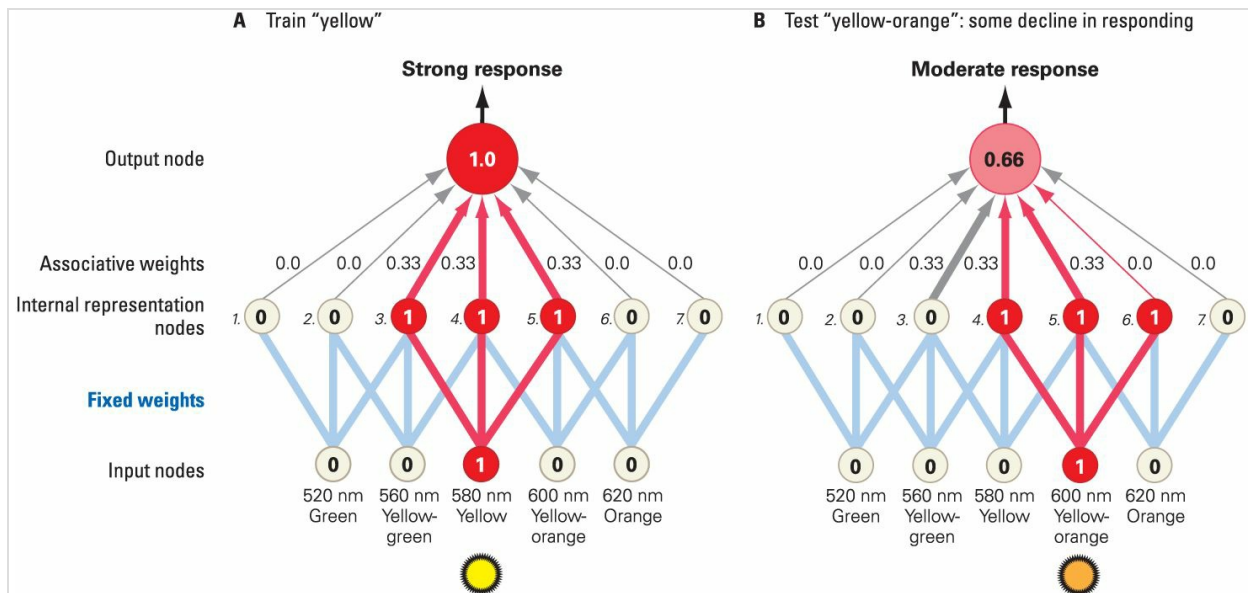
As noted in [Chapter 1](#), ideas much like this were promoted by W. K. Estes in his *stimulus sampling theory* and by David Rumelhart and colleagues in their *connectionist models*. All these approaches embraced the basic idea of [distributed representations](#), in which stimuli are represented by overlapping sets of nodes or stimulus elements. Similarity emerges naturally from

the fact that two similar stimuli (such as yellow and orange lights, or golden retrievers and cocker spaniels) activate elements belonging to both sets. Thus, what is learned about one stimulus will tend to transfer or generalize to other stimuli that activate some of the same nodes.

distributed representation

A representation in which information is coded as a pattern of activation distributed across many different nodes.

Figure 6.4A shows how distributed representations might be used in a network model that is only slightly more complicated than the model of [Figure 6.2](#). This network has three layers of nodes and two layers of weights; by convention, we refer to it as a two-layer network, counting the number of layers of weights. In the network model in [Figure 6.4A](#), each stimulus activates an *input node* that is connected, by a layer of fixed (nonmodifiable) weights, to several nodes in an *internal representation*. These fixed weights, which do not change during learning, are drawn in light blue (except when activated) to help distinguish them from the modifiable weights, which are shown in gray (except when activated). The internal representation nodes are then connected, via the layer of modifiable weights, which will change during learning, to a final *output node*.



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FIGURE 6.4 A network model using distributed representations (A) Presentation of a colored light activates a unique node in the input layer of the network. Each input-layer node is connected, through fixed (nonmodifiable) weights—shown in light blue—to several nodes in the internal representation. The seven nodes in the internal representation (numbered 1 to 7) are then connected by modifiable weights (shown in gray) to a single output node. This particular network has been trained to respond to yellow light. Presentation of the stimulus activates the corresponding input node, which activates three nodes in the internal representation (3, 4, 5), which in turn connect to the output node. If the yellow light is repeatedly paired

with a reward, weights from these three active internal-representation nodes to the output node are strengthened. (B) Later, the network is tested with a similar stimulus (a yellow-orange light) that activates internal nodes 4, 5, and 6. Because yellow-orange and yellow share two overlapping internal representation nodes (4 and 5), some response activation is produced at the output node.

Description

Diagrams A and B have five input nodes each. A circle with a number inside represents each input node. All the input nodes are placed side by side in a row. The input nodes from left to right are as follows. 520-nanometers green. 560-nanometers yellow-green. 580-nanometers yellow. 600-nanometers yellow-orange. And 620-nanometers orange. An output node (represented by a circle with a number inside) is above the third input node circle. Internal representation nodes are represented by a row of circles between the input and output nodes. It has 7 circles numbered 1 to 7 from left to right. Circles 2 to 6 are vertically above the five input nodes.

Fixed weights are solid lines that connect input nodes to the internal representation nodes. Each input node has three fixed weights. These three fixed weights connect each input node to the internal representation node vertically above it, to its immediate left, and to its immediate right. Associative weights are arrows which point from the internal representation nodes to the output node. In diagram A, train yellow, the input node for yellow is numbered 1 and is a red circle. All other input nodes are numbered 0. The three fixed weights for the yellow node are red. All other fixed weights are light blue. The number inside the circles for internal representation nodes numbered, 3, 4, and 5, is 1. These circles are red. All other internal representation node circles have the number 0 inside. The modifiable weight arrows for internal representation nodes 3, 4, and 5 are red thick arrows labeled, 0.33. All other modifiable weight arrows are thin, gray, and labeled, 0.0.

The output node circle is numbered 1.0 and is red. An upward arrow from output node points to the text, strong response. In diagram B, Test yellow-orange: some decline in responding, the input node for yellow-orange is numbered 1 and is a red circle. All other input nodes are numbered 0. The three fixed weights for the yellow-orange node are red. All other fixed weights are light blue. The number inside the circles for internal representation nodes numbered, 4, 5, and 6, is 1. These three circles are red. All other internal representation node circles have the number 0 inside. The associative weight arrows for internal representation nodes 4 and 5 are red thick arrows labeled, 0.33. The modifiable weight arrow for internal representation node 3 is a gray thick arrow labeled 0.33. The modifiable weight arrow for internal representation node 6 is a red thin arrow labeled 0.0. All other modifiable weight arrows are thin, gray, and labeled, 0.0. The output node circle is numbered 0.66 and is light-red. An upward arrow from output node points to the text, moderate response.

Thus, the presentation of a yellow light would activate the “yellow” node in the input layer, which in turn would activate three nodes at the internal representation (nodes 3, 4, and 5); but notice that two of these internal representation nodes (3 and 4) could also be activated by a yellow-green light, and nodes 4 and 5 could also be activated by yellow-orange light. In this manner, yellow-green, yellow, and yellow-orange all activate overlapping sets of internal representation nodes.

The order of nodes in [Figure 6.4A](#) forms a topographic representation, meaning that nodes responding to physically similar stimuli, such as yellow and yellow-orange light, are placed next to each other in the model (a concept introduced in [Chapter 3](#)). Thus, in [Figure 6.4A](#), there is more overlap between the representations for yellow and yellow-orange than between the representations for yellow and orange. There is no overlap between the representations of very different colors, such as green and orange.

Now suppose this network model is trained to respond to a yellow light, which activates the three internal representation nodes, as shown in [Figure 6.4A](#). Note that this network transforms a representation of yellow light in the input row to a different representation in the middle row. The representation of yellow at the input nodes is a discrete-component representation because each light has one and only one node that is activated (and no other colors activate this node). In contrast, the representation at the middle nodes is a distributed representation because the yellow color's representation is distributed over three nodes (3, 4, and 5). So this network can be viewed as one that converts a discrete representation at the input nodes to a distributed representation at the middle nodes. As you will see next, this distributed representation allows the model to account for similarity and generalization.

One way to determine how much the modifiable associative weights should be increased is to use the Rescorla–Wagner learning rule. As presented in [Chapter 4](#), this rule states that weights should be changed in proportion to the error on each trial (with “error” defined as the mismatch between the output that did or did not occur and the correct, or desired, output, which is determined by the actual outcome). Following many trials of training in which yellow is paired with reward, the weights from the three active internal representation nodes would each come to equal 0.33, as shown in [Figure 6.4A](#), where the lines connecting internal representation nodes 3, 4, and 5 to the output node have been thickened. Now when presentation of a yellow light activates nodes 3, 4, and 5, the result would be a net response activation of 1.0 in the output node—that being the sum of these three weights. (Note that the weights from internal representation nodes that have never been activated or associated with reward remain at their initial value of 0.0.)

Compare the distributed network in [Figure 6.4A](#), which was trained to respond to a yellow light, with the discrete-component network in [Figure 6.2](#), which was trained with the very same

stimulus and outcome pairings. In the distributed network of [Figure 6.4A](#), the learning is distributed over weights from three internal representation nodes, each with a trained weight of 0.33; in contrast, the discrete-component network in [Figure 6.2](#) localizes this same respond-to-yellow rule into a single weight of 1.0 from one input node. Both network models give a response of 1.0 when the original yellow light is presented.

The difference between the distributed network of [Figure 6.4](#) and the discrete-component network of [Figure 6.2](#) becomes apparent only on presentation of stimuli that are similar—but not identical—to the trained stimulus. The distributed network is able to generalize. This generalization behavior can be assessed by testing a yellow-orange light, as shown in [Figure 6.4B](#). Here, yellow-orange activates an internal representation that has considerable overlap with the representation activated by the trained yellow light. Specifically, both nodes 4 and 5 are also activated by the yellow-orange light, and each of these internal representation nodes will contribute to partially activate the output node. As a result, a reasonably strong output node activation of 0.66 results, proportional to the two-thirds degree of overlap between the representations for yellow and yellow-orange light. If the same network were tested with orange light, there would be less overlap with the representation of yellow light and a consequently even weaker response.

[Figure 6.5](#) shows that when this model is used to test generalization to a series of novel lights, it produces a stimulus-generalization gradient that decreases smoothly for stimuli of increasing distance from the trained stimulus, similar to the pigeons' generalization gradient shown in [Figure 6.1](#). This ability to capture animals' and humans' natural tendency to generalize—that is, to treat similar stimuli similarly—contrasts markedly with the (lack of a) generalization gradient in [Figure 6.3](#), which was produced using the network that had only discrete-component representation. Thus, even though the two models can learn the same initial task (respond to yellow light), they differ considerably in their generalization performance.

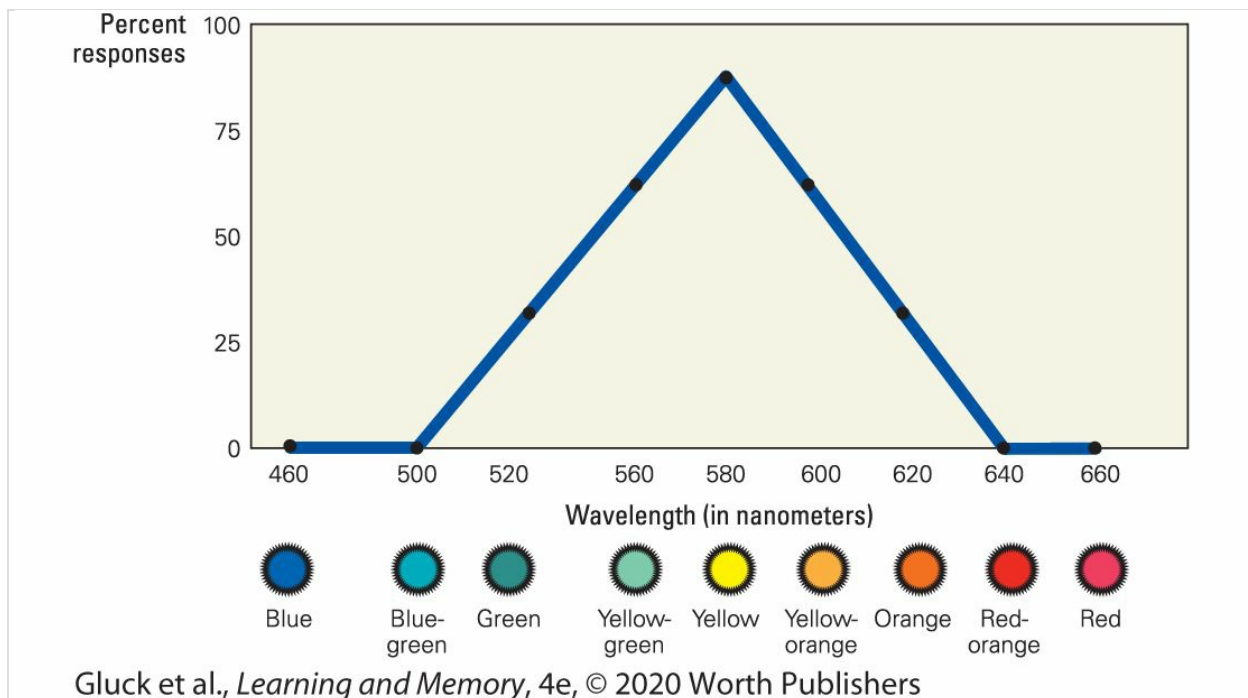


FIGURE 6.5 Stimulus-generalization gradient produced by the distributed-representation model of Figure 6.4 The trained network in Figure 6.4 generates this stimulus-generalization gradient, which shows peak responding to the trained stimulus (yellow) and decreased responses for stimuli that are increasingly different from the trained stimulus. This smooth gradient is similar in shape to the ones seen in animals (including humans), such as that obtained from pigeons, shown in Figure 6.1.

Description

The vertical axis is labeled percent responses, ranging from 0 to 100, in increments of 25. The horizontal axis is labeled wavelength (in nanometers), ranging from 460 to 660, in increments of 20. The wavelength (in nanometers) of blue light is 460, blue-green light is 500, green light is 520, yellow-green light is 560, yellow light is 580, yellow-orange light is 600, orange light is 620, red-orange light is 640, and red is 660. A line starts at (0, 460) and remains constant until (500, 0). It then increases through (530, 30) and (550, 60), before reach a maximum at (580, 80). The line then decreases through (590, 60) and (610, 30) to (640, 0). It then remains constant before ending at (660, 0). All values are estimated.

The generalization gradients in Figures 6.5 and 6.1 are not identical. The network model responds more strongly to orange light than the pigeons do. The exact width and shape of the generalization gradient produced by a model can, however, be manipulated by varying the number of nodes and the amount of overlap of nodes in the model. Nevertheless, the overall shape makes the necessary point. Distributed-representation systems capture a fundamental property of learning: all other things being equal, humans and other organisms tend to treat similar events similarly and to expect similar stimuli to have similar consequences. As you will

read in [Section 6.2](#), these early theoretical notions of shared elements and distributed representations within psychological models presciently anticipated later findings by neuroscientists demonstrating that, indeed, there are overlapping neural networks in our brains for similar stimuli.

TEST YOUR KNOWLEDGE

Discrete-Component or Distributed-Representation Models?

Which of the following learning experiments can be understood and modeled using a discrete-component representation of the stimuli? Which would require a distributed representation with overlapping patterns of elements? ([Answers appear at the end of the chapter.](#))

1. A low-frequency tone predicts a shock, and a high-frequency tone predicts a shock, but a light predicts food. What does a medium-frequency tone most likely predict?
2. Patients in a hospital that has the walls painted blue take 2 days, on average, to recover and be discharged, whereas patients in a hospital that has red walls take 4 days to recover and be discharged. How many days would you expect it to take for patients in a hospital with green walls to recover? Why?

Discrimination Learning and Stimulus Control: When Similar Stimuli Predict Different Outcomes

Remember Sabrina, the fussy 2-year-old from the beginning of this chapter? She is faced with a problem similar to that of Guttman and Kalish's pigeons. If green broccoli tastes awful to her, she may assume that a similar-shaped vegetable, such as cauliflower, has a good chance of being equally unappealing to her. On the basis of this prediction, she may decide it is not worth the risk of experiencing another icky taste to eat the cauliflower... *unless* she is really hungry and there are few other options for her at that meal. If hunger motivates Sabrina to take a chance on the cauliflower, and she finds that she loves the taste, she now has new information: broccoli tastes nasty to her, but cauliflower tastes yummy. From this experience, Sabrina has learned that it is important to *discriminate* between broccoli and cauliflower, even though they have similar shapes (but different colors), because they lead to different outcomes when eaten.

One way of thinking about what has happened when Sabrina discriminates between the nasty-tasting broccoli (which she avoids) and the yummy cauliflower (which she eats) is that Sabrina's

eating behavior has come under the control of external stimuli. Psychologists would say that her behavior has come under [stimulus control](#), in that her choice of what she does or does not eat is now under the influence of the broccoli and cauliflower. Stimulus control is seen in all facets of life. If you drive through an intersection when you see a yellow light but brake to a stop when you see a red light, your own driving behavior can be considered to be controlled, or at least influenced, by these visual stimuli.

stimulus control

The influence of cues in the world on an organism's behavior.

Through experience and discrimination learning, people can master many fine distinctions, as described in the [Chapter 3](#) discussion of perceptual learning. Some can discriminate the tastes of Pepsi and Coca-Cola or can differentiate the french fries at McDonald's from those at Burger King. Similarly, gem cutters may learn to distinguish diamonds of subtly different color or quality, and parents of identical twins learn to tell their children apart. A dog fancier can identify many different breeds of terriers, all of which may appear virtually identical in the eyes of a cat fancier who couldn't care less. Two objects or substances that initially seem very similar, or even indistinguishable, eventually come to be distinguishable when each is repeatedly paired with a different label, name, or outcome.



Constantin Iosif/Shutterstock

Can you taste the difference between Pepsi and Coca-Cola? If not, how might you train yourself to do so?

What determines whether two stimuli are to be treated as similar (generalization) or different (discrimination)? This is a fundamental problem in psychology, and the ensuing discussion reviews some of what psychologists have learned about it from years of careful studies of generalization in animals and humans. For a look at how stimulus control and discrimination learning can be applied to help you sleep better, see the [“Learning and Memory in Everyday Life”](#) box.

LEARNING AND MEMORY IN EVERYDAY LIFE

Sleep Better Through Stimulus Control

Have trouble falling asleep at night? Wake up tired and then find yourself unable to keep awake during the psychology exam review session? Maybe what you need is a different set of stimulus cues controlling your behavior in your bedroom. One reason many people have trouble sleeping at night is that their bed and bedroom are filled with stimulus cues that have been associated with wakeful activities, not with sleeping. Do you watch TV in your bedroom? If so, then the sight of the TV (even off) may ready your brain to watch TV instead of sleep. Do you lie in bed for hours trying to fall asleep, worrying about your exam (or the guy who did not return your last seven email messages)? If so, then the bed and the room are becoming associated with—and hence controlling—the initiation of your worrying behaviors, not your sleep behaviors.

Sleep experts routinely advise good sleep hygiene—which mostly boils down to limiting your bedroom to stimulus cues associated with sleep and only sleep. (Sex in the bedroom is an exception to this rule; it is an activity that actually promotes sleep.) Because discrimination training is rarely accomplished in one trial (except in the case of fear associations), it may take many days or weeks to train yourself to have better sleep habits. The first step, however, is to identify all the stimulus cues that lead to (and, hence, control) wakeful behaviors—the laptop, tablet, smartphone, and TV—and move them into another room.

Discrimination Learning and Learned Specificity

We turn now to look more closely at discrimination learning and what it tells us about mechanisms for learning and generalization. One important study in this area, conducted by Herbert Jenkins, a Canadian psychologist, was based on the behavior of two groups of pigeons. One group received standard training in which a 1,000-Hz tone signaled that pecking a key would result in food delivery ([Jenkins & Harrison, 1962](#)). Because the birds received the food only after pecking the key, the food reinforcement was contingent on the birds' behavior. Recall from [Chapter 5](#) that this operant conditioning can be described as

S (1,000-Hz tone) → R (key peck) → O (food)

The second group of pigeons received discrimination training in which one of two different (but similar) stimuli was presented on each trial. For these pigeons, the 1,000-Hz tone signaled that a key peck would result in food reinforcement, but another, very similar tone of 950 Hz signaled that a key peck would *not* result in food reinforcement:

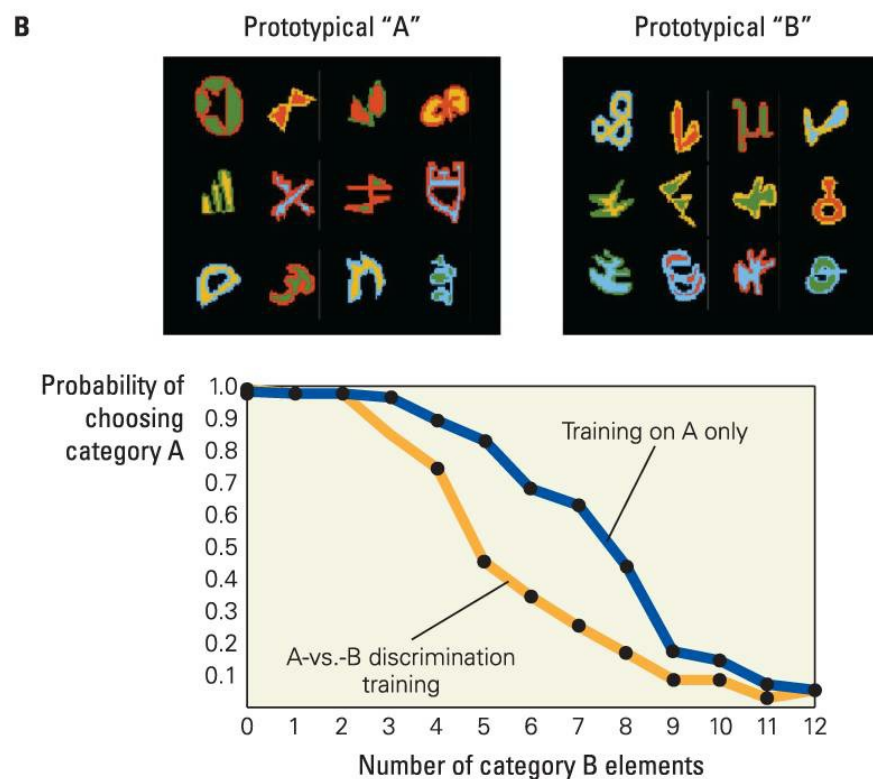
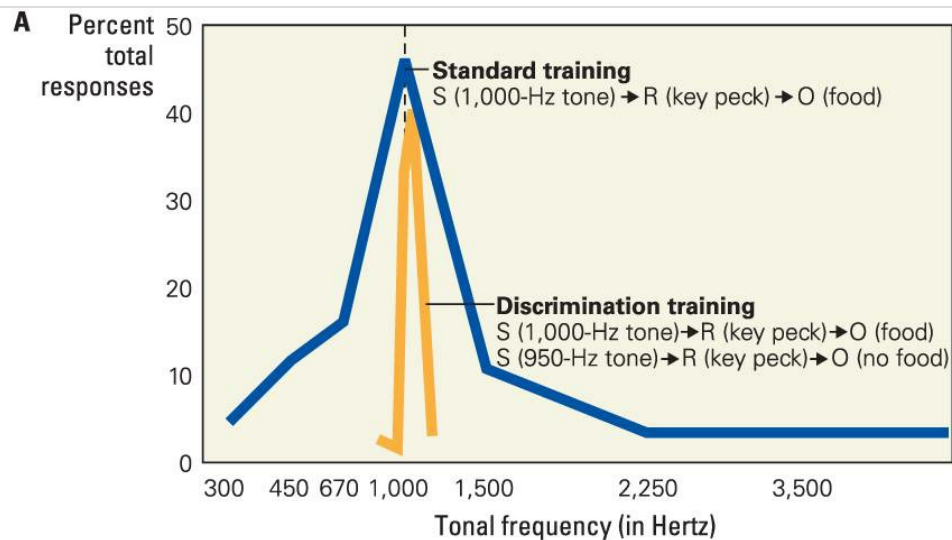
S (1,000-Hz tone) → R (key peck) → O (food)

S (950-Hz tone) → R (key peck) → O (no food)

Because the two stimuli in this experiment differ within a single dimension (namely, tone frequency), the paradigm is referred to as an *intradimensional discrimination*. In contrast, many of the paradigms presented in earlier chapters, where animals learned to discriminate between stimuli that differ across multiple dimensions (such as tones and lights), are known as *extradimensional discriminations*.

Following this training, both groups of pigeons were given test trials with new tones, ranging from very low-frequency (300-Hz) tones to very high-frequency (3,500-Hz) tones. The

experimenters measured how frequently the birds pecked in response to each test tone. As shown in [Figure 6.6A](#), the group that received training with a single tone (“standard training”) showed a broad, bell-shaped generalization gradient similar to what Guttman and Kalish found for light stimuli (see [Figure 6.1](#)).



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B: Catherine E. Myers

FIGURE 6.6 Generalization gradients following discrimination training (A) Data from a study in which one group of pigeons learned by “standard training” that a 1,000-Hz tone signaled that key pecks would be reinforced; these birds showed a typical generalization gradient when tested with novel tones. A second group of pigeons learned that the 1,000-Hz tone signaled that pecks would be rewarded but that the (very similar) 950-Hz tone signaled no reward (“discrimination training”); these birds showed a much steeper generalization gradient. (B) Examples of the prototypical category A and category B exemplars used in the [Wills and McLaren \(1997\)](#) categorization study and a graph showing generalization gradients following discrimination versus A-only training.

(A) Data from [Jenkins & Harrison, 1962](#). (B) Research from [Wills & McLaren, 1997](#).

Description

Graph A has a vertical axis labeled, percent total responses, ranging from 0 to 50, in increments of 10. The horizontal axis is labeled, tonal frequency (in hertz), with markings at 300, 450, 670, 1,000, 1,500, 2,250, and 3,500. A line labeled, standard tuning, starts at (300, 4), increases to (670, 15), and further increases to a maximum at (1,000, 45). The line then decreases to (1,500, 10), and further decreases to (2,250, 3). Beyond 2,250 hertz, the line is constant at 3 responses. Standard training has a stimulation S (1,000-hertz tone), response R (key peck), which then results in output O (food). A line labeled, discrimination training, starts at (950, 2), decreases to (1,000, 1), then increases to a maximum at (1,100, 40) through (1,000, 35). It then decreases to end at (1,300, 3). Discrimination training has a stimulation S (1,000-hertz tone), response R (key peck), which then results in output O (food). Discrimination training also has another stimulation S (950-hertz tone), response R (key peck), which then results in output O (no food). Graph B has a vertical axis labeled, probability of choosing category A, ranging from 0 to 1, in increments of 0.1. The horizontal axis is labeled, number of category B elements, ranging from 0 to 12, in increments of 1. A line labeled, training on A only starts at (0, 1), and passes through (1, 0.99), (2, 0.98), (3, 0.97), (4, 0.9), (5, 0.8), (6, 0.65), (7, 0.6), (8, 0.4), (9, 0.2), (10, 0.15), and (11, 0.1), before ending at (12, 0.05). A line labeled, A versus B discrimination training starts at (0, 1), and passes through (1, 0.99), (2, 0.98), (4, 0.75), (5, 0.45), (6, 0.35), (7, 0.25), (8, 0.15), (9, 0.06), (10, 0.06), and (11, 0.01), before ending at (12, 0.05). Accompanying illustration shows two squares titled, prototypical A and prototypical B, each with a set of 12-member abstract geometric elements, arranged in 3 rows of 4 each. The shapes of the elements in each square are different. The elements in prototypical B have a unique set of 12 abstract geometric elements when compared to prototypical A.

In contrast, the pigeons that underwent discrimination training showed a different pattern of generalization. Their generalization gradient was much steeper, centered immediately around the 1,000-Hz tone and dropping off much more rapidly—so that they were not responding at all to the nearby 950-Hz tone or to any other tones higher or lower than 1,000 Hz. The difference in shape of the two generalization gradients in [Figure 6.6A](#) shows that, with discrimination training, the generalization gradient can change to reflect more specific (i.e., more narrowly focused) responding to the reinforcing stimulus. In fact, the generalization gradient following discrimination training in [Figure 6.6A](#) looks very much like the gradient in [Figure 6.3](#), which illustrated what a component-representation model predicts when there is no overlap in stimulus representations. This suggests that [Figure 6.3](#) was not wrong; it was just an illustration of what

happens after discrimination training when the stimuli do not have overlapping representations.

A similar difference between the generalization gradients that emerge from training with a single stimulus as compared to discrimination training can be seen in a study of human category learning in which people were shown visual stimuli consisting of sets of abstract geometric elements ([Wills & McLaren, 1997](#)). Each subject was presented with two 12-member sets of abstract geometric elements. One of the sets was presented as a prototypical exemplar of category A, and a distinct set of 12 was presented as prototypical of category B ([Figure 6.6B](#)). Other, potential exemplars were then presented, constructed with different mixtures of the elements from the prototypical A and the prototypical B exemplars. An exemplar was considered to be a member of category A if it possessed at least 8 out of the 12 elements from the category A model.

Participants in this study were assigned to one of two types of training. Participants who received discrimination training were shown many examples of category A and many examples of category B, with each example labeled according to its correct category assignment. Other participants did not receive discrimination training but instead were only shown exemplars from category A. After receiving one or the other form of training, participants were given a subsequent generalization test in which they were asked to judge whether unlabeled exemplars were members of category A. As you can see from the generalization gradients in [Figure 6.6B](#), people who were trained only with the category A stimulus showed broader generalization gradients than those who received A-versus-B discrimination training, endorsing more of the ambiguous exemplars as being members of category A. This pattern of data echoes the pattern seen in Herbert Jenkins's pigeon study ([Figure 6.6A](#)): broad generalization gradients after standard acquisition training and narrower, more focused generalization gradients after discrimination training.

Errorless Discrimination Learning and Easy-to-Hard Transfer

[Chapter 4](#) lays out the principles of error-correction learning, where learning in the Rescorla–Wagner model is driven by prediction errors—that is, by the mistakes in prediction that an animal makes when it does or does not anticipate an unconditioned stimulus (US). [Chapter 4](#) also reveals that errors need not be the key to all forms of learning. In fact, as we will soon see, it is

possible to develop training regimens that all but avoid errors—and, in doing so, to enable animals (including people) to learn very difficult discriminations with little or none of the frustration and emotional angst that come from making mistakes (emotions you might have experienced yourself on the last test or quiz you took).

Herbert Terrace developed a training procedure he called **errorless discrimination learning**. Training begins with a discrimination task that is readily learned and then transitions to a similar but different discrimination task that is harder to learn so as to eliminate, or drastically reduce, the errors made during the harder task.

errorless discrimination learning

A training procedure in which a difficult discrimination is learned by starting with an easy version of the task and proceeding to incrementally harder versions as the easier ones are mastered.

In one study of errorless discrimination learning, Terrace trained pigeons to discriminate between a red disk (S+) and a green disk (S-) and peck only the red disks ([Terrace, 1963](#)). Normally, this would take many trials and produce many errors, much as in the classical and operant conditioning curves shown in [Chapters 4](#) and [5](#). Terrace got the pigeons to learn this discrimination quickly, and with almost no errors, by giving them a hint—like a set of “training wheels” for learning—to keep them from making the wrong choices. Early in training, he made the red disks very bright and bold (to naturally attract the pigeons’ attention) and kept them available for 3 full minutes. In contrast, the green disks were presented as dark or dimly lit and were available for only a few seconds. Since pigeons generally do not peck at dark objects, especially if they have limited time to do so, the pigeons rarely pecked the wrong green S- keys. Gradually, over many trials, Terrace increased the brightness and duration of the green S- keys until they were as bright and lasted as long as the red S+ keys. Terrace’s pigeons were now able to discriminate the two colors without the extra hint and guidance of the modified duration or brightness of the keys.

Terrace’s errorless learning procedure has had many applications in education but has seen its greatest success in training people with learning disabilities. By means of a procedure similar to the one in the Terrace study, children with Down syndrome were taught to identify basic shapes such as ovals and rectangles ([Duffy & Wishart, 1987](#)). This procedure involved flash cards that initially contained only the correct shape for the name given (so no errors were possible). Then the children were shown cards containing two choices, one being the correct shape in a large

format and the other being the incorrect shape set very small. The very small size of the incorrect choice guided the child to ignore it, akin to the effect of dimming the lights in the Terrace study; thus, the child was naturally inclined to choose the larger option. Over time, the sizes of the two alternative choices were made increasingly similar until they were the same (much as Terrace gradually made the lights for the pigeons the same brightness and duration), and the children with Down syndrome ultimately learned to identify shapes correctly—avoiding the errors and frustration that would have resulted had they been given the final test at the very start.

Errorless discrimination learning does have significant drawbacks, however. While it produces rapid and strong (and painless) learning of the discriminations being trained, many later studies showed that this learning is very rigid and inflexible, adhering to the details of the original training procedure rather than generalizing to new situations and stimuli not trained in this fashion ([Clare, Wilson, Carter, Roth, & Hodges, 2002](#); [Jones & Eayrs, 1992](#)). Nevertheless, in the study described above, the errorless discrimination learning, by preventing errors, also prevented the frustration, trauma, and self-doubt the children with learning disabilities might have suffered from making many errors at the start. Like a parent who doesn't want to see a child fall and hurt himself trying to ride a two-wheeler before he is ready, the errorless learning procedure offers a gradual form of learning that begins with an easy task and then incrementally moves to the harder version when the person is ready and able.



Ariel Skelley/The Image Bank/Getty Images

Falling off a two-wheeler can hurt, so this child's parents adopted a variation of what learning procedure?

Beyond Similarity

So far in this chapter, we have discussed the process of learning in three of the cases schematized in [Table 6.1](#): (1) those in which similar stimuli lead to similar consequences; (2) those in which dissimilar stimuli lead to different consequences; and (3) those in which similar stimuli lead to different consequences. But there is a fourth possibility: two dissimilar stimuli might predict the same outcome. As you will see next, generalization can occur even when physical similarities between the stimuli are absent.

One way in which generalization can extend to dissimilar stimuli is through frequent pairing, or co-occurrence. [William James \(1890\)](#) argued that if all cold objects were wet and all wet objects were cold, the two characteristics would be viewed as a single concept; the presence of one (e.g., wet) would imply the presence of the other (i.e., cold). James concluded that organisms would tend to cluster, or treat equivalently, stimulus features that tend to co-occur. More recently, in his historical novel *The Alienist* (1994), author Caleb Carr told the story of a nineteenth-century forensic psychologist—a former student of William James at Harvard—who used this very example from James’s Introductory Psychology lectures to hunt for a serial murderer who was terrorizing New York City. The criminal had a childhood history of being beaten by the people he loved; as a consequence, he came to view violence and love as so intertwined that when one was present, he expected or exhibited the other, beating to death any woman he loved and loving any woman whom he beat.

Much common wisdom is based on examples of this kind of clustering, such as “where there’s smoke, there’s fire.” The same kind of clustering of stimuli based on common outcomes or consequences can be demonstrated in the learning laboratory, as seen in the following discussions of sensory preconditioning and acquired equivalence.

Sensory Preconditioning: Co-occurrence and Stimulus Generalization

One way in which co-occurrence of two stimuli can lead to generalization is through a training procedure known as [sensory preconditioning](#), in which the prior presentation of two stimuli together, as a compound, results in a later tendency for any learning about one of these stimuli to generalize to the other.

sensory preconditioning

Training in which presentation of two stimuli together as a compound results in a later tendency to generalize what is known about one of these stimuli to the other.

In the laboratory, sensory preconditioning is usually tested in three phases, as summarized in [Table 6.2](#). In phase 1, animals in the compound exposure group (i.e., the experimental group) are first exposed to a compound of two stimuli, such as a tone and a light presented simultaneously. In phase 2, the animals learn that one of the stimuli by itself (such as a light) predicts an important consequence (such as a blink-evoking airpuff), and they eventually give a blink response to the light. In phase 3, the animals are exposed to the tone only.

TABLE 6.2 Sensory preconditioning

Group	Phase 1	Phase 2	Phase 3: test
Compound exposure	<i>Tone + light (together)</i>	Light → airpuff → blink!	Tone → blink!
Separate exposure (control group)	<i>Tone, light (separately)</i>	Light → airpuff → blink!	Tone → no blink

Take a look at the training of the compound exposure group in sensory preconditioning in [Table 6.2](#), and you might find that it resembles the blocking procedure from [Table 4.5](#)—only backwards! This reversal—training the compound stimulus tone + light first (as in sensory preconditioning) rather than last (as in blocking)—has a big impact on what is or isn't learned. It demonstrates a key aspect of the error-correction nature of learning: that the order of presentation matters.

Most of the compound exposure group will show at least some response to the tone. In contrast, a second (control) group of animals are given exposure to the tone and light separately in phase 1. They are then given phase 2 training identical to the phase 2 training of the other group: they learn that the light by itself predicts the airpuff. But when tested with the tone alone in phase 3, these animals show little or no response ([Thompson, 1972](#)).

It seems that for the experimental group, the compound exposure in phase 1 establishes an association between the tone and light. In phase 2, the light becomes associated with the airpuff, and this learning is indirectly transferred to the tone, too. This transfer can be interpreted as an *outcome-based generalization* because the tone and light are assumed to be followed by the same outcome (i.e., they both predict the airpuff) even though they do not have any relevant physical similarity. An outcome-based generalization can be contrasted with a *similarity-based*

generalization, which arises naturally between two stimuli that are physically similar. Sensory preconditioning shows that co-occurrence of two stimuli is sufficient to produce outcome-based generalization from one stimulus to the other.

As suggested above, the order of events (phase 1 first versus phase 2 first) is very important. Reversing the order would produce a blocking procedure (as in [Table 4.5](#)), a paradigm that leads to the opposite effect—little or no conditioning to the tone even when it is repeatedly presented in training.

Acquired Equivalence: Novel Similar Predictions Based on Prior Similar Consequences

Another form of outcome-based generalization can occur when two noncombined stimuli share the same consequence—that is, predict the same outcome. In this case, it is possible for generalization to occur between two very dissimilar stimuli even if they never co-occur. Consider two girls, Mandy and Kamila, who look nothing alike but who both love hamsters. Later, you learn that Mandy’s favorite fish is a guppy. Given that Mandy and Kamila have similar taste in pet rodents, you might expect that Kamila will also like Mandy’s favorite pet fish.

Geoffrey Hall and colleagues have found a similar form of generalization in their studies with pigeons ([Bonardi, Rey, Richmond, & Hall, 1993](#); [Hall, Ray, & Bonardi, 1993](#)). In one study, they trained pigeons to peck at a light that alternated among six different colors, identified as A1, A2, B1, B2, X1, and Y1. The researchers then trained the animals to associate the four two-color sequences A1–X1, A2–X1, B1–Y1, and B2–Y1 with the arrival of a food reward ([Table 6.3](#), left column).

TABLE 6.3 Acquired equivalence

Phase 1 training	Phase 2 training	Phase 3: test
A1 → X1 → food	A1 → food	A2: strong pecking response
A2 → X1 → food		
B1 → Y1 → food	B1 → no food	B2: no strong response
B2 → Y1 → food		

In effect, the colors A1 and A2 were “equivalent” because they were both paired with X1. Likewise, colors B1 and B2 were equivalent in their pairing with Y1. Next, the pigeons learned that pecking to A1 alone resulted in food; in contrast, no food followed pecks to B1 alone ([Table 6.3](#), middle column). In phase 3, the pigeons were tested for response to A2 and B2. The birds responded strongly to A2 but not to B2 ([Table 6.3](#), right column), suggesting that the birds had learned equivalencies in phase 1 between A1 and A2 and between B1 and B2. After phase 1 training that A2 was “equivalent” to A1 and phase 2 training that responses to A1 resulted in food, the birds expected that responses to A2 would also result in food.

Hall and colleagues called this behavior [acquired equivalence](#) because prior training that two stimuli were equivalent increased the amount of generalization between them—even if those stimuli were superficially dissimilar. Similar effects have been shown in humans ([Spiker, 1956](#)) and rats ([Hall & Honey, 1989](#); [Honey & Hall, 1991](#)). In summary, although physical similarity is a frequent cause of generalization, generalization may also have other causes. Animals and people can learn to generalize from one stimulus to another that is superficially dissimilar if the stimuli have a history of co-occurring or of predicting the same consequence.

acquired equivalence

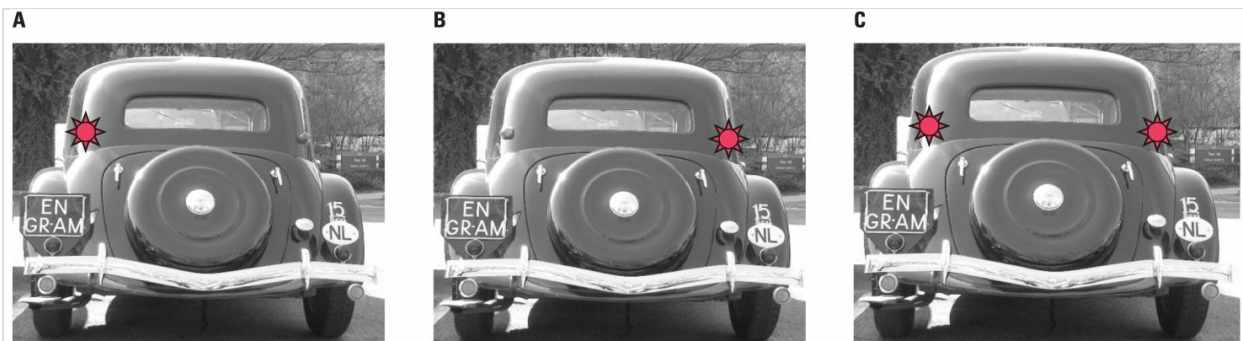
A learning and generalization paradigm in which prior training in stimulus equivalence increases the amount of generalization between two stimuli, even if those stimuli are superficially dissimilar.

Negative Patterning: When the Whole Means Something Different Than the Parts

In another complication of generalization from compounds composed of stimuli with different attributes, consider what happens when Sabrina starts playing with other children. She might learn from experience on the playground that freckle-faced children are usually bratty and that red-haired children are also bratty. Given these two associations, you might expect her to be especially wary of a girl she meets one day who is both red haired and freckle faced. Based on her past experiences, she expects the girl to be really horrid because she has both of the attributes Sabrina associates with brattiness. All other things being equal, we, like Sabrina, tend to assume that combinations of cues will have consequences that combine, and possibly summate, what is known about the individual cues.

But is this assumption always correct? What if a certain combination of cues implies something

totally different from what the individual cues mean? For example, you know what the letter “c” sounds like, and you know what the letter “h” sounds like, but when you see these two letters together as “ch,” you know that they represent quite a different sound. Thus, readers of English learn that certain combinations of letters can sound very different from their component letters. Consider another example, involving driving a car. While waiting at an intersection, you notice that the antique Citroën car in front of you has its *left* rear red taillight flashing, signaling the car is about to turn left ([Figure 6.7A](#)). If its *right* rear red taillight were flashing, you would assume the driver intends to turn right ([Figure 6.7B](#)).



Mark A. Gluck

FIGURE 6.7 The challenge of interpreting combined cues (A) Left blinking light means left turn, (B) right blinking light means right turn, (C) both lights blinking means that the driver has turned on the hazard lights.

Description

Photo A shows a red star on the left taillight. Photo B shows a red star on the right taillight. Photo C shows red stars on the right and left taillights.

But what if *both* taillights are flashing, as in [Figure 6.7C](#)? Although either the left or the right turn signal flashing indicates an imminent turn, both taillights flashing together certainly does not indicate a combination of two turns. Instead, twin flashing taillights signal a hazard: the car is proceeding slowly or is disabled. Although our tendency may be to assume that what is true of component features presented individually is also true of their combination, clearly it is possible to override this tendency to generalize from components to compounds, as do automobile drivers who learn that the combination of both left and right lights flashing means something quite different than either of them flashing alone.

This kind of situation, where cue combinations have radically different meanings than their

components, has been extensively studied with both animal and human learning tasks. Suppose, for example, that a rabbit in a classical conditioning study is trained to expect that either a tone or a light, presented alone, predicts an airpuff US to the eye but that there will be *no* airpuff US if the tone and light appear together. To respond appropriately, the animal must learn to respond with a blink to the individual tone cue or light cue but to withhold responding to the compound tone-and-light cue. This task, schematized as

Tone → airpuff US

Light → airpuff US

Tone + light → no US

is known as **negative patterning** because the response to the individual cues is positive, whereas the response to the compound (i.e., the “pattern”) is negative (no response).

negative patterning

A behavioral paradigm in which the appropriate response to individual cues is positive, whereas the appropriate response to their combination (pattern) is negative (no response).

Because both the tone and the light cues are part of the tone-and-light compound, there is a natural tendency for the animal to generalize from the component cues to the compound cue and vice versa. However, this natural tendency to generalize from component features to compounds does not work in this case because the components and the compound are associated with very different outcomes.

With training, the negative-patterning task can be mastered by many animals, including humans. **Figure 6.8** shows an example of negative patterning in rabbit eyeblink conditioning ([Kehoe, 1988](#)). After only a few blocks of training, the animals learn to give strong responses both to the tone alone and to the light alone. In addition, during this early phase of training, the rabbits make the mistake of overgeneralizing from the components to the compound, giving incorrect strong responses to the compound tone-and-light stimulus (which is not followed by an airpuff). Only with extensive further training do the rabbits begin to suppress responding to the tone-and-light compound.

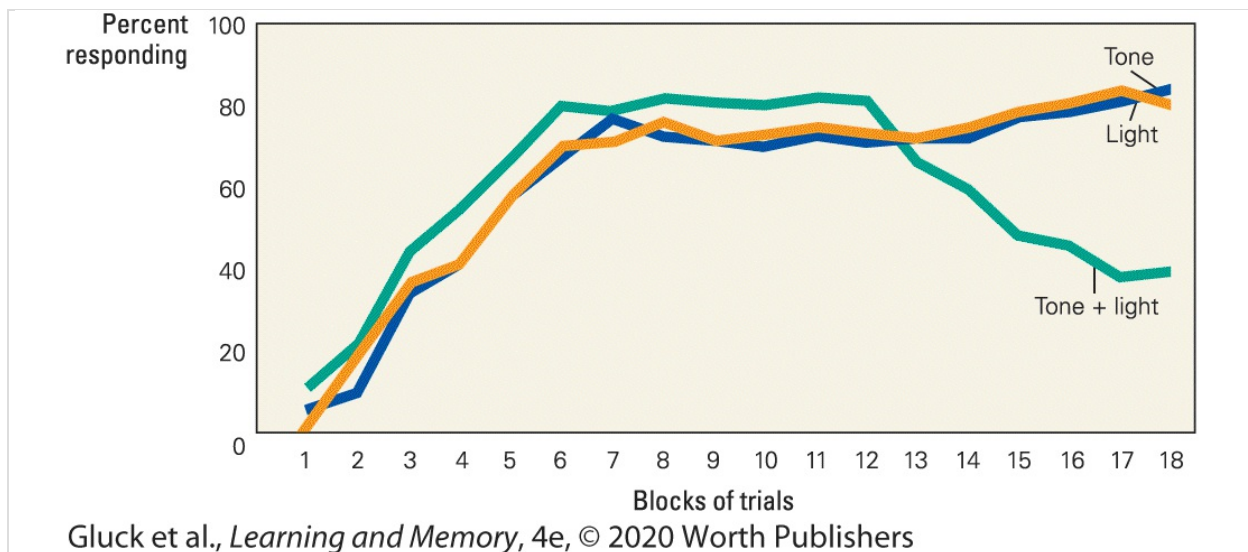


FIGURE 6.8 Negative patterning in rabbit eyeblink conditioning Negative patterning requires learning to respond to two cues (a tone and light) when each is presented separately but to withhold the response when the two cues are presented together.

Data from [Kehoe, 1988](#).

Description

The horizontal axis is labeled blocks of trials, ranging from 0 to 10, in increments of 1. The vertical axis is labeled percent responding, ranging from 0 to 100, in increments of 25. A line labeled tone starts at (1, 5) and rises through (2, 10), (3, 35), (4, 40), (7, 80), (10, 70), (11, 75), and (14, 75), before ending at (18, 85). A line labeled light starts at (1, 0) and rises through (3, 35), (4, 40), (6, 75), (8, 80), (9, 75), (13, 75), and (17, 80), before ending at (18, 70). A line labeled tone plus light starts at (1, 10) and rises through (2, 20), (3, 50), (6, 80), (7, 75), (8, 80), (12, 75), (13, 60), (14, 55), (15, 40), (16, 39), and (17, 35), before ending at (18, 40). All values are estimated.

Can associative-network learning models such as those illustrated in [Figure 6.2](#) (with discrete-component representation) or [Figure 6.4](#) (with distributed representations to indicate stimulus similarity) provide an explanation of how rabbits and other animals learn negative patterning? [Figure 6.9](#) illustrates why single-layer network models, like those in [Figure 6.2](#), that use discrete-component representations cannot learn the negative-patterning problem. To produce correct responding to the tone alone, the modifiable associative weight from the input unit encoding tone must be strengthened to 1.0 ([Figure 6.9A](#)). To produce correct responding to the light alone, the weight from the input unit encoding light must also be strengthened to 1.0 ([Figure 6.9B](#)). But this means that if the tone and light are presented together, activation will both flow through those modifiable weighted connections and produce strong responding to the compound—stronger responding, in fact, than to either component alone ([Figure 6.9C](#)).

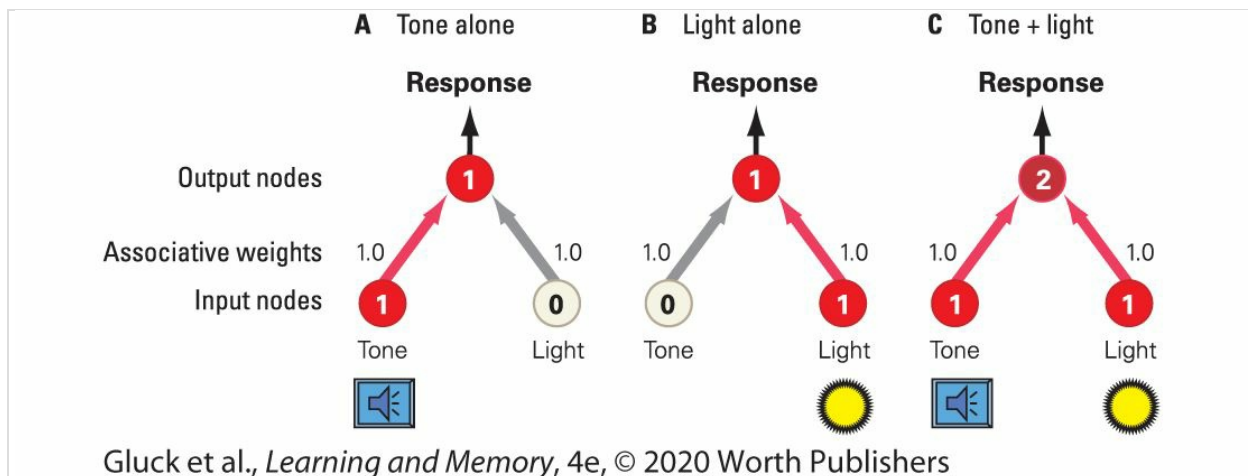


FIGURE 6.9 Failure of a single-layer network with discrete-component representations to learn negative patterning (A) For the tone cue to correctly generate a strong response, the connection from that input to the output must be strongly weighted. (B) For the light cue to correctly generate a strong response, the connection from that input to the output must also be strongly weighted. (C) Consequently, when both tone and light cues are present, the network will incorrectly give a strong response.

Description

All three diagrams have two input nodes each. Each input node is represented by a circle with a number inside. The input nodes are placed side by side in a row. The input nodes from left to right are tone and light. An output node (represented by a circle with a number inside) is above the space between the two input nodes. Associative weights are arrows which point from the input nodes to the output nodes. In diagram A, tone alone, the input node for tone is numbered 1 and is a red circle. The input node for light is numbered 0. The associative weight arrow for input node tone is a red thick arrow labeled, 1.0. The associative weight arrow for input node light is a gray thick arrow labeled, 1.0. In diagram B, light alone, the input node for light is numbered 1 and is a red circle. The input node for tone is numbered 0. The associative weight arrow for input node light is a red thick arrow labeled, 1.0. The associative weight arrow for input node tone is a gray thick arrow labeled, 1.0. In diagrams A and B, the output node circle is numbered 1 and is red. An upward arrow from output node points to the text, response. In diagram C, tone plus light, both the input nodes are numbered 1 and are red circles. The associative weight arrows for the input nodes are red thick arrows labeled, 1.0. The output node circle is numbered 2 and is red. An upward arrow from output node points to the text, response.

All the weights could be decreased, of course, to reduce the level of response to the compound in [Figure 6.9C](#), but this would also, incorrectly, reduce responding to the individual components. In fact, there is no way to assign associative weights in the network of [Figure 6.9](#) that would make the network respond correctly to all three different types of training trials.

Negative patterning is just one example of a larger class of learning phenomena that involve configurations of stimuli and that cannot be explained using single-layer networks. To master

such tasks, an animal must be sensitive to the unique configurations (or combinations) of the stimulus cues, above and beyond what it knows about the individual stimulus components. Solving such problems requires more complex networks than the single-layer network of [Figure 6.9](#)—either a single-layer network with more complex nodes that are sensitive to configurations of combinations of cues or a network with multiple layers of weights and nodes that can learn complex cue combinations.

TEST YOUR KNOWLEDGE

Discriminating Between Generalization Paradigms

To be sure that you have learned to discriminate between the various behavioral paradigms of generalization, see if you can assign each of the following four paradigms to the real-world example that best exemplifies it in the numbered list below.

(Answers appear at the end of the chapter.)

- a. Discrimination training
 - b. Sensory preconditioning
 - c. Acquired equivalence
 - d. Negative patterning
-
- 1. Elizabeth is quite impressed by men who, on a first date, bring her either candy or flowers. However, if a man shows up with both, she is turned off, feeling he is coming on too strong.
 - 2. As a child, Samson learned that people who have deep voices also tend to have beards. He later became convinced that men with beards are strong, and he inferred that a deep voice is also likely a sign of strength.
 - 3. By playing snippets of music by Brahms, then Schubert, and then Brahms again, a music teacher is able to teach his class how to recognize the style of each.
 - 4. Mark and Kaori enjoy many of the same foods and people. Based on this observation, Mark guesses that Kaori will also like a particular song of which he is fond.

Variability in Training and the Generalization of Knowledge and Skills

When not studying psychology, Stephanie has a side career as an aspiring classical pianist. She spends many hours a week practicing at home on the baby grand piano that her parents bought for her. Made by the Steinway & Sons piano company, it is one of the finest pianos you can buy. Although Stephanie could do all her practicing on this wonderful piano in the comfort of her own home, she chooses instead to expand her routine to include regular practice on the Bösendorfer (another fine piano) in the university's music department. In addition, she sometimes goes to the student lounge to practice on the smaller Yamaha upright piano there.

With such a wonderful piano at home, why does Stephanie go to such effort to practice on these other pianos as well? The reason is that Stephanie often travels to other cities to compete in competitions for young pianists and, of course, does not bring along her own piano. Rather, she must play on whatever piano is available at the competition venue. These instruments might come from different manufacturers, each with a different feel and some with different-size keyboards. If Stephanie practiced only on her own Steinway at home, her skills might become tied to, and dependent on, the individual idiosyncrasies of that one piano. This could result in her having trouble playing on different pianos at the competitions. To ensure that her practice generalizes to as many different types of pianos as possible, Stephanie purposefully introduces variability into her training routine. She wants to ensure that no matter what the subtle differences between the pianos (a case of dissimilar stimuli), she will be able to produce an outcome that her audience will enjoy.

Experimental studies of learning and generalization have indeed confirmed that variability in training can improve generalization. However, studies of people solving mathematical word problems suggest that this variability is especially helpful in cases like that of the pianist Stephanie, where the person has a high level of initial knowledge and skill ([Braithwaite & Goldstone, 2015](#)). In contrast, for those with very little initial knowledge, it helps to start out with minimal variation. Accordingly, when Stephanie was a little girl just learning to play piano, she took all her classes on her teacher's piano. Only later in life, as she became more expert, did she introduce the schedule of variability in training that she now follows to prepare for competitions.

Concept Formation, Category Learning, and Prototypes

Before we can delve further into the topic of concepts and categories, it is important to clarify the distinction between the two. [Concepts](#) are internal psychological, or mental, entities; they are ideas we construct on the basis of our experiences with the world. In contrast, [categories](#) are a feature of the world—classes or divisions of people or things that have some shared observable characteristics ([Smith, 1989](#)). Part of the confusion that occurs between concepts and categories is that we often use the same word to refer to both. The category “dog” is a subdivision of mammals and refers to all entities in the real world that can be described as dogs. The concept

“dog,” however, is the mental representation through which we perceive, understand, and think about the category dogs.

concept

An internal psychological representation of a real or abstract entity.

category

A division or class of entities in the world.

The formation of concepts is a basic cognitive process through which people organize, describe, and generalize about the world. It is the means by which we learn about categories in the world. In turn, categories help us make inferences about objects and events and guide us in predicting the future. Forming concepts requires both generalization and discrimination. We generalize within a class but discriminate between its members and also between different classes. To understand the concept of dogs, we must recognize a wide variety of dogs, be able to generalize the concept of dog to new dogs that we have never seen before, and be able to discriminate between dogs and other similar entities, such as wolves and coyotes.

How and why do psychologists differentiate between concept formation (learning about categories) and discrimination learning (learning different responses to different stimuli)? The distinctions between the two are subtle, and many learning paradigms can be accurately described as either one. In general, however, discrimination learning refers to paradigms in which a relatively small number of stimuli are considered (sometimes only two), the stimuli are simple idealized and often unnatural images or sounds produced in a laboratory, and the distinction between positive and negative examples is usually well defined. In contrast, concept formation often involves many (often countless) stimuli, which may be naturalistic and highly varying and complex, and the distinction between positive and negative instances of a concept may be ill defined or very difficult to make. Although concept formation was originally viewed as belonging exclusively to the domain of human learning, while discrimination learning was predominantly the purview of animal learning, this distinction no longer holds, as you will see in the material to follow.

In the remainder of [Section 6.1](#), we will discuss how concepts can emerge from discrimination training, we will introduce the concept of prototypes in understanding and reasoning about natural categories, and we will note some of the generalization errors that can result from reasoning about categories of people.

Emergence of Concepts Through Discrimination Learning

Animals have remarkable abilities of discrimination learning. Pigeons have been trained to discriminate between the Baroque classical music of Bach and the modern twentieth-century neoclassical music of Stravinsky ([Porter & Neuringer, 1984](#)). Not only were the pigeons able to learn this discrimination well, they were able to generalize this learning to novel music from other composers who worked in related stylistic genres. For example, when presented with the music of Telemann, another Baroque composer, the birds pecked the Bach response disk; upon hearing the mid-twentieth-century neoclassical music of Elliott Carter, they pecked the Stravinsky disk. It seems from these studies that pigeons are able to form and distinguish the abstract concepts of Baroque and neoclassical music styles.

Pigeons have also learned to discriminate between the abstract paintings of Picasso and the impressionist paintings of Monet and then to generalize this learning to other paintings by these and other artists they had never seen before ([Watanabe, Sakamoto, & Wakita, 1995](#)). The pigeons were first trained to peck an illuminated lamp without any painting stimulus. Half the birds were then randomly assigned to the Picasso+ group, and their responses to the Picasso (but not Monet) paintings were reinforced by a 4-second access to a feeder containing hemp seeds. The other half of the birds were assigned to the Monet+ group and were reinforced only when they pecked Monet paintings.



Nano Calvo/AGE Fotostock

If a pigeon can learn to differentiate the impressionists from the abstract painters, can you? How might you learn to do it if you can't already?

The pigeons learned this discrimination quite easily. Much as the pigeons in the music study appeared to infer the concepts of Baroque and neoclassical styles, these pigeons appear to have induced the distinction between impressionist and abstract painting styles. That is, after being trained on only Picasso and Monet paintings, the pigeons identified the paintings of other impressionists like Renoir and Cezanne as being “like” those of Monet, while paintings by other abstract painters, such as Matisse and Braque, were treated like Picasso paintings ([Figure 6.10](#)). Interestingly, the birds in the painting study were successful at recognizing the novel abstract paintings of Picasso even when they were shown upside down. However, the pigeons did much less well when the more realistic and representational paintings of Monet were presented to them upside down.



Shigeru Watanabe

FIGURE 6.10 Pigeon learning to discriminate between abstract and impressionist paintings

Prototypes and the Structure of Natural Categories

The above descriptions of categories and concepts have mostly referred to situations, often contrived in a laboratory experiment, in which membership in a category was all or none: either an entity did or did not belong to a given category. Natural categories encountered in real life, however, can be much more difficult to determine. Are robins and penguins both equally good examples of birds? Most people would say no. Is an olive a fruit? Well, technically, yes, but it is not what we usually think of when speaking of fruit, and relatively few people would classify it that way. In a series of seminal papers, [Eleanor Rosch \(1973, 1975\)](#) showed that natural categories often have ill-defined boundaries, with some members of categories being viewed as better, more central, and more typical examples of the category than others. Robins, for example, are generally viewed as typical members of the class of birds, whereas penguins are not.

If I ask you to imagine a typical bird, the creature you think of might not look like any particular breed of bird, but it probably has the physical features typically associated with birds—wings, feathers, a beak, and two legs—and functional attributes such as singing, flying, and perching in

trees. This kind of abstract representation of the idealized or typical member of a category is called the category's **prototype**. (Remember the prototypical A and prototypical B exemplars in [Figure 6.6](#)?) Rosch argued that the most commonly used concepts are organized around prototypes that are based on family resemblances, sets of features shared by most members of a category. Thus, a robin would be judged a very typical bird because it has many features in common with most other birds, whereas a penguin has relatively few features shared with other birds ([Rosch, Mervis, Gray, Johnson, & Boyes-Braem, 1976](#)).



Top: Dmytro Pylypenko/
Shutterstock;
bottom: Jozef Sowa/
Shutterstock

Are these both typical birds? If not, what does that tell us about our concept of birds?

prototype

The central tendency or idealized version of a category.

Categories with a high degree of family resemblance (and hence many shared clusters of common features) are especially useful because they permit **inductive inference**, the making of logical inferences that are probably (but not necessarily) true and are usually based on attempts to draw a general rule from one or more specific instances or premises ([Goldstone, Kersten, & Carvalho, 2012, 2017](#)). If we know something is a dog, then it probably barks, has four legs, eats dog food, and likes to mark its territory using trees, fire hydrants, and occasionally people's legs. Of course, there are exceptions to all these inferences, such as the Basenji dog of Africa, which does not bark, and the rare dog that has had the misfortune to lose a limb. While it is true that very few dogs will attempt to leave their scent on you if no tree is available, it is still prudent to move out of the way when any dog standing next to you lifts its leg.

inductive inference

A logical inference that is probably (but not necessarily) true and is usually based on attempts to draw a general rule from one or more specific instances or premises.

Inductive inferences are about what is likely (but not necessarily) true; based on those inductive inferences, we make choices about how we want to respond. From this perspective, concepts derived from categories are tools for helping us induce meaningful relationships in the world. They facilitate our making choices that advance our own interests (e.g., keeping our shoes free of dog urine). But even as we form useful concepts about categories in the world, we may find that the inductive inferences we make from them can sometimes lead us astray.

Stereotypes, Discrimination, and Racism in Generalizations About Other People

When our behavior toward another person is based on that person's membership in a category defined by race, ethnicity, national origin, gender, religion, age, or some other identifying feature, we are implicitly making a generalization on the basis of a **stereotype**, a set of beliefs about the attributes of the members of a group ([Ashmore & Del Boca, 1981](#)). Many stereotypes conform roughly to statistical realities, but others are founded on false images of what a typical

member of a group is thought to be like: the soccer-loving Englishman, the punctual German, the amorous Frenchman, the tall and loud American (or Texan). Stereotypes can be benign; they can be useful; or they can lead unfairly to prejudicial discrimination.

stereotype

A set of beliefs about the attributes of the members of a group.

In the context of the psychological study of learning, the term *discrimination* usually refers to the ability to tell apart two or more stimuli and generate different responses to each. In everyday social contexts, however, *discrimination* is more often used to mean the unfair differential treatment of individuals on the basis of the group to which they are perceived to belong. Stereotypes have a long history of being associated with a broad range of social evils and used to promote many harmful and illegal forms of discriminatory treatment ([Jussim, 2012](#); [Steele & Aronson, 1995](#)). A critical question for this discussion is: When does *appropriate* psychological discrimination become *inappropriate* social discrimination against an individual?

As you read earlier in this chapter, categorization is a basic cognitive process through which we form concepts to organize, describe, and understand the world around us. Forming conceptual categories allows people, as well as all other animals, to recognize statistical patterns in our world that help predict future events. Generalization is the natural (and rational) extension of these processes. Unfortunately, when generalizations are made about a group of people, there is a fine, and hotly debated, line between what constitutes a rational generalization and what is an offensive misrepresentation inviting prejudicial behavior toward members of that group.

How Do People Learn Stereotypes?

Although some of our stereotypes are based on personal encounters with members of another group, many stereotypes are socially communicated through our parents, friends, and secondhand reports in books, movies, TV, and the Internet, all of which vary greatly in their accuracy. (For more on social learning, see [Chapter 11](#).) In the process of acquiring stereotypes, people filter what they attend to through the personal lens of their own needs and self-interest. We tend to believe good things about the groups we belong to and view groups that are different from ours with varying degrees of skepticism.

Once we have formed a negative stereotype about members of another group, we are more likely

to pay attention to examples that confirm this stereotype. This process is known as [confirmation bias](#) ([Pettigrew, 1979](#)). For example, a business owner might continue for years to do fine by hiring only people who have a Harvard degree, on the assumption that graduates of a state university would be inferior employees. As a result, the business owner would never learn the really important and useful fact that he could have done better if he had hired the brilliant *summa cum laude* from the local state university whom he did not even bother to interview. We only learn about what (and whom) we pay attention to.

confirmation bias

A tendency to ignore information that conflicts with a prior belief and focus on information that is consistent with that belief.

How Do People Interpret and Use Stereotypes?

In the aftermath of attacks on several people by pit bulls, many communities enacted laws that ban pit bulls, arguing that such laws are justified because *pit bulls are dangerous dogs*. Others objected to these laws, calling them a form of “speciesism” because, they claimed, these laws were unfairly biased against the many pit bulls that have never harmed anyone at all ([Schauer, 2003](#)). Is the stereotype of pit bulls as dangerous dogs accurate or not?

Although seemingly clear in meaning, this generalization about pit bulls is actually ambiguous, in that logically it has three potential interpretations. Do we mean that (1) all pit bulls are dangerous, (2) most pit bulls are dangerous, or (3) pit bulls are more dangerous than most other breeds of dog? According to the first interpretation, the existence of even a single non-dangerous pit bull would imply the stereotype is inaccurate. According to the second interpretation, a randomly chosen pit bull is more likely to be dangerous than not because half or more of all pit bulls are dangerous. Although either of these interpretations of “pit bulls are dangerous” is logically plausible, neither corresponds to what is generally believed by people who hold a stereotype about a category (whether of people or dogs).



Yvonne Hemsey/Hulton Archive/
Getty Images

What do people know about pit bulls? What do they believe about them, and why?

In fact, the interpretation most in line with psychological research about generalizations is the third: *pit bulls are more dangerous than other breeds of dog*. Under this interpretation, a generalization about pit bulls as dangerous could be accurate even if only 10% of pit bulls are dangerous, so long as this is higher than the percentage of dogs of other breeds that are dangerous (which, for example, might be only 2% of dogs overall). “Pit bulls are dangerous dogs” could be a useful stereotype if knowing that a dog is a pit bull produces an appropriate increase—by five times, in this hypothetical example—in our expectation that the dog will be dangerous.

This discussion demonstrates that our concept of pit bulls, like our concept of most natural categories, is fuzzy and ill defined. The relationships between the category (pit bull) and some of the key features or attributes ascribed to its members (e.g., being dangerous) are only partial or probabilistic. In other words, certain features may be common to many of the members but not to all of them, as is seen in Rosch’s work on natural categories and their prototypes, described earlier.

The concept of stereotype is similar to that of prototype but with subtle differences worth noting.

A prototype is usually defined *externally* based on the central or common tendencies of exemplars of the category, while a stereotype is a *psychological concept* that may or may not closely correspond to an accurate prototype for the category of people (or dogs) to which it refers. Rosch argued that natural categories are adopted when they provide us with value in improving our ability to make inductive inferences ([Rosch et al., 1976](#)). We adopt stereotypes in the belief that they do the same. In some ways, prejudicial behaviors toward individuals—or dogs—can be understood as resulting from an unconscious cost–benefit comparison: the natural mechanisms operating in the brain may decide that the cost of finding out about an individual person or dog is too high relative to the ease of making a choice informed by only an inductive inference based on category membership.

All animals, including humans, are sensitive to the informational value of cues for organizing stimuli that pour in from the world around them ([Corter & Gluck, 1992](#); [Gluck & Bower, 1988](#)). This sensitivity suggests that people are more likely to form new categories, defined by certain features, when those features can be easily perceived (as is the case with race, ethnicity, and gender) and when the people judge the categories to be potentially predictive of other features of interest to them, such as an unknown person’s income level, criminal propensities, or amount of education—information that may not be so readily perceivable in each new individual they meet. Forming categories is the brain’s way of connecting perceivable cues and inferred attributes. When the connections are false, however, such categorizations can lead to inaccurate predictions or expectations about inferred attributes.

How Can We Distinguish Between Appropriate and Inappropriate Uses of Stereotypes?

Stereotypes, like all generalizations, enable us to apply prior experience to new situations when we have little or no specific information available. However, using stereotypes appropriately requires finding a balance between specificity (does the stereotype apply only to members of a particular group?) and generality (does it apply to all members of that group?). Many commonly held assumptions about specific groups of people may accurately reflect statistical realities ([Jussim, 2012](#)). For example, it is a fact of life in the United States today that different racial groups have statistically different crime rates, medical maladies, educational achievements, and incomes. But it is important to remember that there will be large numbers of individuals in a group who do not fit the statistical profile. Moreover, these statistics are not static but can evolve

and change with time. Even if a stereotype was once accurate, it may no longer be so.

Accurate generalizations, including stereotypes about people, can be put to beneficial use as long as they are interpreted correctly as descriptors of statistical characteristics of a large group. But even a fundamentally accurate generalization—a stereotype that reflects some statistical reality in the world—can form the basis for inaccurate conclusions if its limitations as a statistical variable are misunderstood, causing it to be applied inflexibly and inappropriately to people's detriment.

Theories and data from the learning laboratory offer no easy solutions to the social problems caused by stereotypes and prejudice, but they do help us understand that the underlying behavior of generalizing to individuals from category membership is not, in and of itself, a bad thing. The trouble with stereotypes arises (1) when people use generalizations that are not based on true or appropriate statistics and (2) when people use statistically valid generalizations to justify discrimination against individuals, denying the possibility that the generalization may not pertain to everyone in the group.

All species, including sea hares, rats, and humans, are hardwired to infer contingencies between cues and outcomes, obtaining information about the makeup of the world that increases their ability to predict the future. Although some forms of stereotyping have negative consequences for individuals and society, it would be overgeneralizing about generalizing to conclude that all forms of pre-judging about groups of other people are inappropriate.

Interim Summary

- Generalization enables us to apply prior experience to new situations. However, it requires finding an appropriate balance between specificity (knowing how narrowly a rule applies) and generality (knowing how broadly the rule applies).
- The problem of generalization is really four different subproblems: (1) what happens when similar stimuli (or events) have the same outcome (or consequences), (2) what happens when they have different outcomes, (3) what happens when dissimilar stimuli have the same outcomes, and (4) what happens when dissimilar stimuli have different outcomes?
- A generalization gradient shows the strength of an organism's expectation that the consequences of one stimulus will follow other, similar stimuli. Through discrimination training, the generalization gradient can be modified, showing that the organism has learned

to distinguish (and respond differently to) highly similar stimuli.

- Different models of generalization represent stimuli in different ways. A discrete-component representation depicts each stimulus (or stimulus feature) as one node (or network component). Such representations apply only to situations in which the similarity between the stimuli is small enough that learning about one stimulus should not transfer appreciably to the others. In contrast, distributed representations, in which stimuli are represented by sets of nodes (that overlap when the stimuli share some of the same features or elements), provide a framework for modeling stimulus similarity and generalization.
- Variability in a training regimen can improve generalization of the ability being trained, especially in individuals with high levels of initial knowledge and skill.
- Categories enable us to make inferences—that is, generalizations—about members of categories and guide us in predicting the future (to the extent possible), leading us to formulate expectations about new experiences, events, or people.
- When our behavior toward another person is based on that person's membership in a category defined by race, ethnicity, national origin, gender, religion, age, or some other identifying feature, we are implicitly making a generalization based on a stereotype. Stereotypes can be harmful when people use them improperly—for example, to justify discriminating against individuals as if the generalization can be assumed to pertain to every individual in the group.

6.2 Brain Substrates

Why devote a whole chapter to generalization and discrimination learning? As noted in the introduction, generalization is the reason for learning. A medical student may be exposed to many rules and examples during her years of training, but there is no way she can study every possible kind of patient that she might ever meet. Rather, as she approaches each new and novel patient in her practice, she will have to decide when and in what ways their particular symptoms and case histories are like and unlike the examples and principles she studied in medical school.

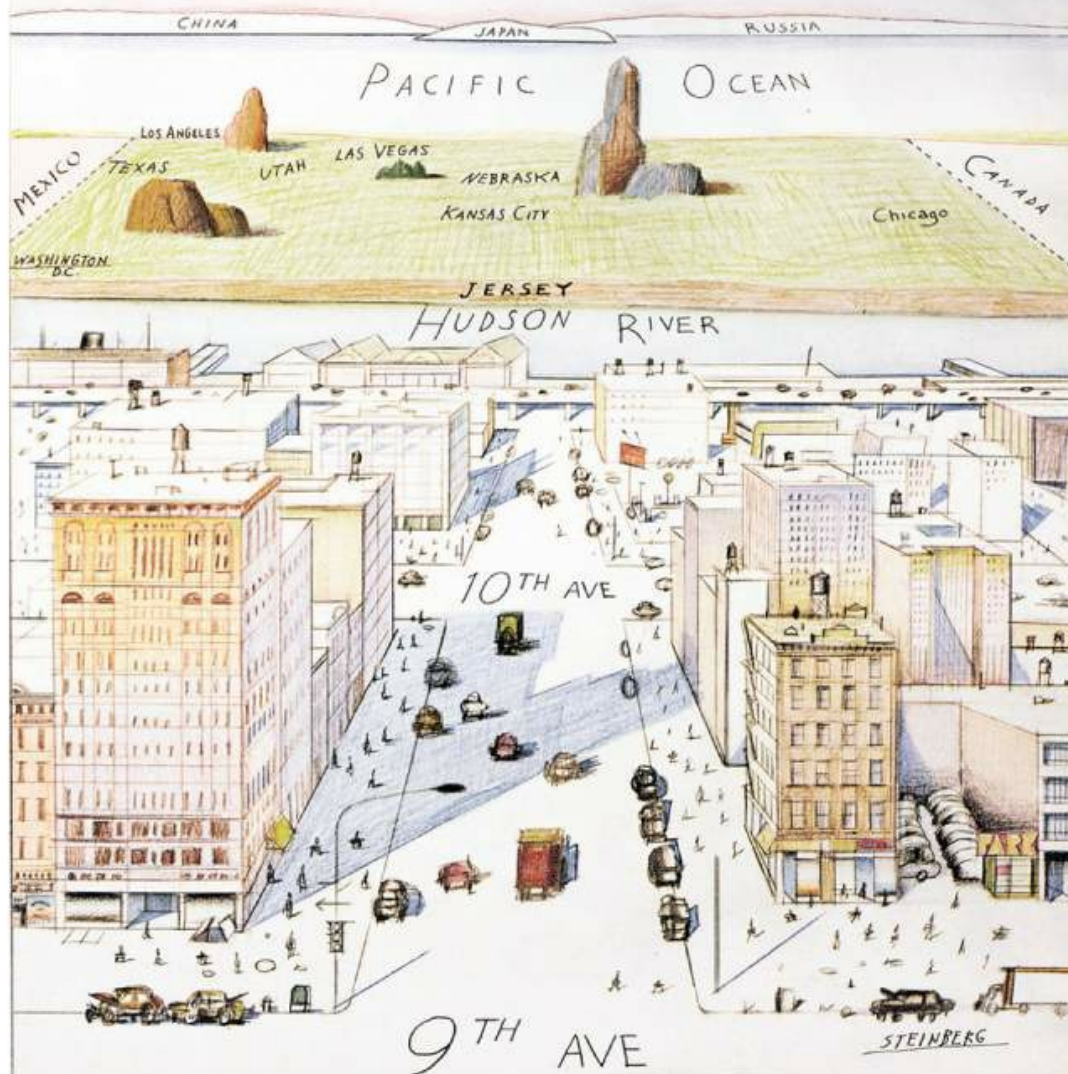
The psychological theories described in the previous section were developed to explain the four kinds of generalization we explored there. These theories were, for many years, just abstractions—tools that psychologists used to help organize, understand, and predict behavioral phenomena. As you read in that section, many of the theories were developed as simple models of how relationships between stimuli and outcomes might be represented—that is, “encoded”—in patterns of mental activity or the structure of associative networks. In recent years, however, the associative network theories from psychology have become increasingly useful to neuroscientists as a blueprint for how the brain might actually accomplish certain mental processes. These developments are the focus of our inquiries in this section.

One of the ideas we’ll be considering is represented by artist Saul Steinberg’s most famous cover for the *New Yorker* magazine ([Figure 6.11](#)), caricaturing his view of a typical New York City dweller’s mental map of the world. Within this depiction of New York City, Ninth and Tenth Avenues are drawn in such fine detail that they take up half the map. The rest of the country, the area between New Jersey and California, is represented as a largely barren desert marked by a few scattered rocks and hills.

Mar.29,1976

THE NEW YORKER

Price 75 cents



Saul Steinberg, "View of the World from 9th Avenue," 1976. © The Saul Steinberg Foundation/ Artists Rights Society (ARS), New York.

FIGURE 6.11 Caricature of a New York City resident's mental map of the United States Regions that are important to a

New Yorker, such as Ninth and Tenth Avenues, are exaggerated in this representation, while other regions, such as the entire Midwest, are disproportionately small.

Description

The bottom half of the cover page shows the ninth and tenth avenues in the New York City connected by a vertical road. Buildings line the sides of the avenues and the road. Many types of vehicles are on the road. Above the visualization of the city is the Hudson River. Above the Hudson River is a rectangular land mass representing Los Angeles, Texas, Washington D C, Utah, Las Vegas, Nebraska, Kansas City, Jersey, and Chicago. The rectangular land mass is bounded on the left by Mexico, right by Canada, and on the top by the Pacific Ocean. At the top of the map, China, Japan, and Russia are depicted as small landmasses.

This illustration satirizes the belief of many New Yorkers that they are living in the most important place in the world. It also illustrates an important psychological principle. Fine distinctions that are meaningful to New Yorkers, such as the differences between fashionable street addresses, are emphasized and highly elaborated in their mental maps; these places are physically spread out and detailed within this drawing. At the same time, distinctions that may be irrelevant to the typical New Yorker, such as the difference between Illinois and Indiana, are de-emphasized and compressed into less space on the map.

The idea of representations of important places being elaborated with fine details and taking up lots of space, while representations of less important places lack definition, reflects a recurring theme in this book. [Chapter 2](#) describes motor and sensory maps in the cortex and how the area in the cortex devoted to each body part relates in extent to the levels of sensitivity and fine motor control of that body part (a topic that appears again in [Chapter 8](#) in the context of cortical maps and how they expand with extensive practice, as seen in violin players). The basic principle explored in these discussions is that regions of the cerebral cortex that mediate a highly practiced skill can expand with practice, while regions that are less used may even shrink.

What does this have to do with generalization? As described by the models in [Section 6.1](#), representations directly influence generalization behaviors. If the drawing in [Figure 6.11](#) were indeed an accurate representation of a New Yorker's mental map of the world, what would that imply? Based on this representation, you might expect a New Yorker to make very few generalizations between two New York neighborhoods despite their proximity. A New Yorker can discriminate between real estate prices on Central Park West (expensive) and those on nearby Columbus Avenue (much cheaper), even though these two streets are actually only a few

hundred yards apart (and even though a Midwesterner would consider both of them expensive to live on). In other words, a New Yorker is unlikely to generalize from one to the other and make the mistake of expecting that an apartment for sale on Central Park West would cost the same as an identical apartment one block over on Columbus Avenue. (But if you find one that does, grab it!)

The preceding example might suggest that New Yorkers are pretty knowledgeable people (and, indeed, they like to think so). However, everything changes when they are tested on their knowledge of the Midwest. For a New Yorker with a mental map like [Figure 6.11](#), all the Midwestern states are functionally equivalent. If you tell this New Yorker that Kansas has lots of corn farms, he will probably assume the same must surely be true of Iowa and Illinois. In other words, a New Yorker who has identical (or very similar) mental representations of the various Midwestern states will automatically generalize from one to another and have great difficulty telling them apart.

Are New Yorkers unusual in having such a warped view of the world, with extreme generalization in some areas and extreme discrimination in others? To some extent, we all create similarly idiosyncratic worldviews, with uniquely distorted representations; distinctions important to us are enhanced, while less relevant ones are de-emphasized. For example, students tasked with sketching a map of the world tend to draw their home region disproportionately large—and in the center of the map. Many American students enlarge North America relative to the other continents. In contrast, European students tend to draw Eurocentric maps, while students from Australia are naturally more likely to place Australia and Asia in the center.

These kinds of representational distortions, although sometimes comic in their egocentricity, are actually very useful. A similar process allows a violinist to devote more of her cerebral cortex to the fine control of her left hand, which must perform the demanding work of holding down different combinations of strings on the proper frets of the instrument, as compared with her right hand, which is only used for the less intricate movements of drawing the bow back and forth across the strings. With only a finite number of neurons available to control all our behaviors, it makes sense to allocate them efficiently according to which distinctions are, and are not, most needed.

The studies reviewed in the first part of this Brain Substrates section relate the plasticity of the sensory cortices to behavioral properties of stimulus generalization and discrimination learning. The section concludes with a look at the special role of the hippocampal region in stimulus generalization.

Cortical Representations and Generalization

[Chapter 3](#) introduces three key findings about perceptual learning and cortical plasticity. First, when perceptual learning occurs in humans, cortical changes accompany the enhanced discrimination abilities. Second, the selectivity of individual cortical neurons in responding to specific stimulus features can be modified through experience. Third, learning can change the spatial organization of interconnected neurons in the sensory cortex. Each of these principles of perceptual learning and cortical plasticity has a bearing on the generalization and discrimination behaviors described in [Section 6.1](#). Keep them in mind while reading the following discussions of the brain mechanisms that contribute to generalization.

Cortical Representations of Sensory Stimuli

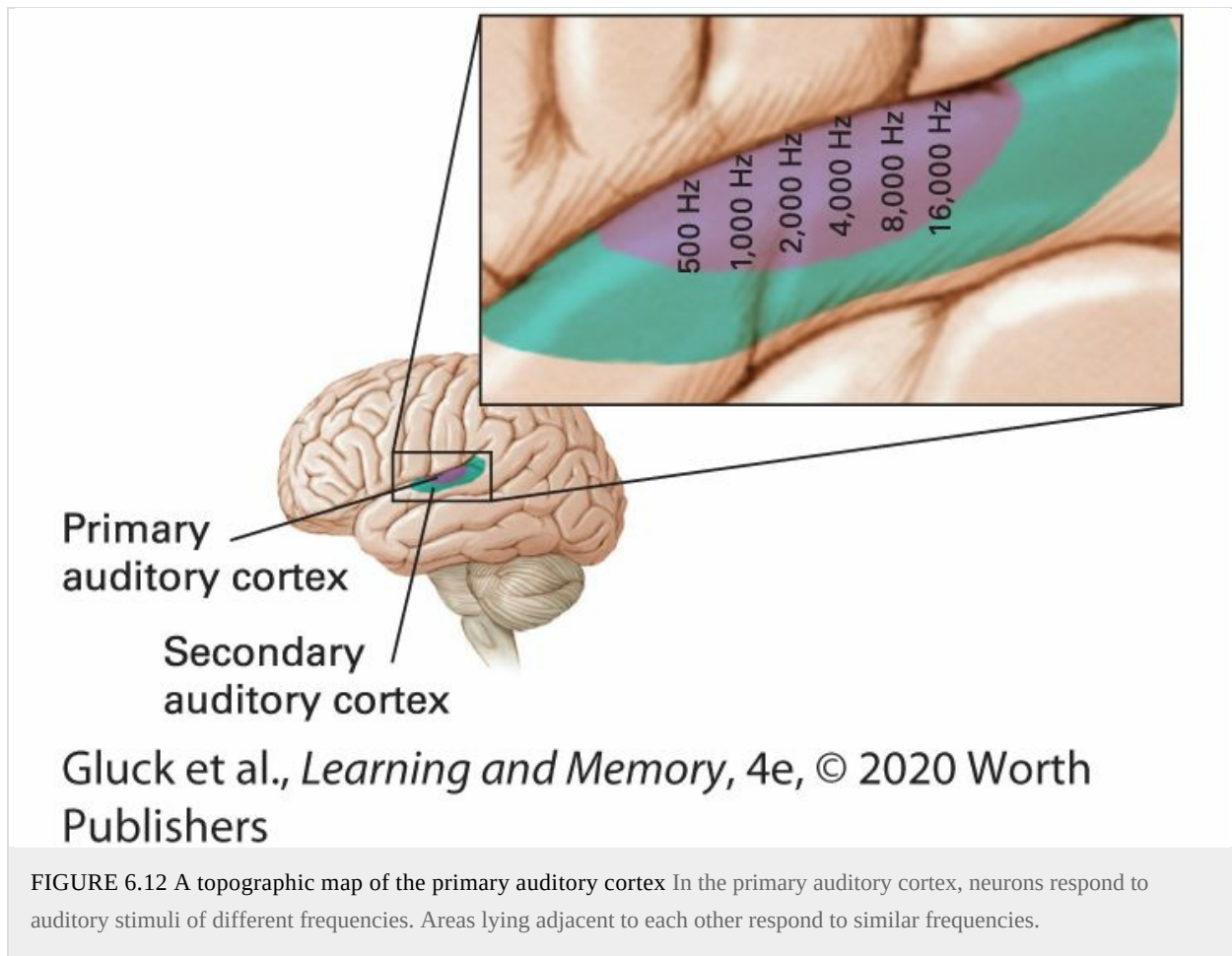
Recall that initial cortical processing of sensory information occurs in a region dedicated to the sensory modality in question: primary visual cortex (or V1) for vision, primary auditory cortex (or A1) for sounds, primary somatosensory cortex (S1) for touch, and so on. From these initial processing regions, sensory information progresses to higher sensory areas that integrate it with other information, first within and then across sensory modalities. Many primary sensory cortical areas are organized topographically (as described in [Chapters 2](#) and [3](#)), which means that each region of the cortex responds preferentially to a particular type of stimulus and that neighboring cortical regions respond to similar stimuli. It is therefore possible to draw various “maps” on the cortical surface by studying the responses of different cortical regions.

For example, as described in [Chapters 2](#) and [3](#), S1 is a thin strip of cortex running down each side of the human brain (see [Figure 3.13](#)). Some neurons in S1 respond primarily to stimulation on a particular finger, some primarily to touch on a certain region of the face, and so on. By stimulating a body part and noting neuronal responses, it is possible to draw a “map” of the body on S1, with each body part in the map lying over the cortical region that shows the greatest response when the corresponding part of the body part is touched, as [Figure 3.13](#) illustrates.

To some extent, adjacent areas within S1 contain neurons that respond to neighboring areas of the body. (There are some discontinuities; for example, [Figure 3.13](#) shows that the parts of S1 that respond to sensations on the fingers lie near those that respond to sensations on the forehead.) Parts of the body that are especially sensitive to touch, such as fingers and lips, activate larger areas of S1. The result is a *homunculus*, a distorted neural representation of the human figure with exaggerated hands and lips but a greatly shrunken torso, as shown in [Figure 3.13](#). This figure of a human is distorted in much the same way as the map on the *New Yorker* cover in [Figure 6.11](#): regions where fine discriminations are important (such as sensations on the fingertips) are disproportionately large and detailed.

The primary somatosensory cortex in other animals shows similar organization, with the homunculus replaced by a distorted figure of the species in question, altered to reflect body areas important to that animal. For instance, primates receive a great deal of touch information through their fingers and lips, which are disproportionately elaborated in their cortical map. Rats receive a great deal of information from the displacement of their whiskers, meaning that a rat's whisker area is disproportionately represented on its cortical map.

The primary auditory cortex (A1) lies near the top of the temporal lobe in humans, and it too is organized as a topographic map, as shown in [Figure 6.12](#). In A1, however, neurons respond to sound rather than to touch stimulation. Areas of A1 that are adjacent to each other respond to similar frequencies. Each neuron in the auditory cortex responds most to one particular tone, as discussed in [Chapter 3](#).



Description

A magnified view of the primary and secondary auditory cortices has the following frequencies listed from left to right on the primary auditory cortex: 500, 1,000, 2,000, 4,000, 8,000, and 16,000 Hertz.

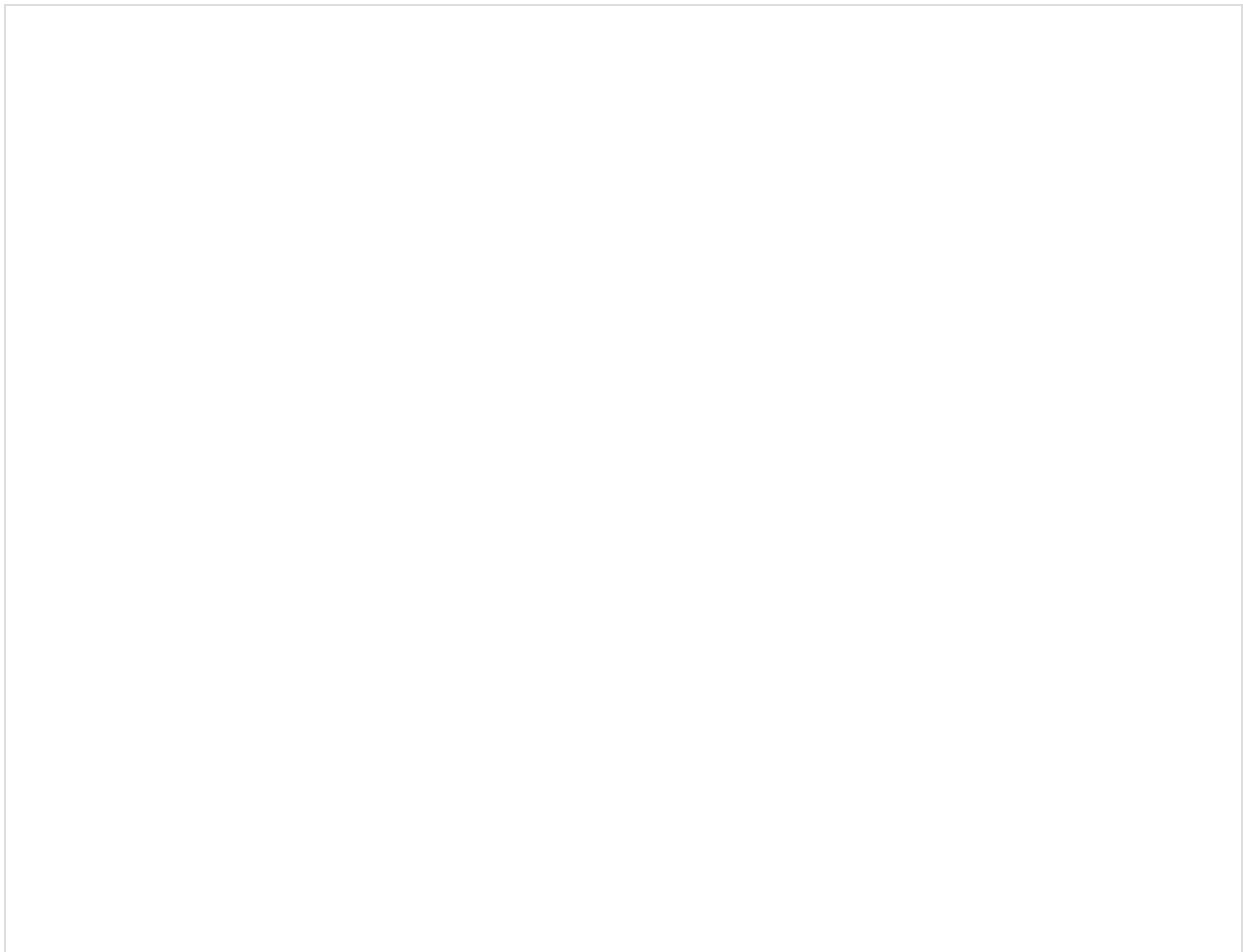
We know what these auditory maps look like because of studies that recorded how often a single neuron fires in response to tones of different frequencies. [Figure 3.12](#) presents data collected from such an electrophysiology experiment. This information represents the receptive field for a neuron, meaning the range (or “field”) of physical stimuli that activate it. The wider a neuron’s receptive field, the broader the range of physical stimuli that will activate the neuron.

Shared-Elements Models of Receptive Fields

How well does the functioning of receptive fields of neurons match the theories of generalization described in [Section 6.1](#)? If the brain is organized to use distributed representations, then physically similar stimuli, such as two tones with similar frequencies, will activate common

nodes, or neurons. In other words, two similar tones—of, say, 550 Hz and 560 Hz—should cause overlapping sets of neurons to fire.

Figure 6.13A shows how brain organization might resemble the distributed-component representation, or “shared-elements” model, from [Section 6.1](#). A 550-Hz tone might activate sensory receptors in the ear that travel to the primary auditory cortex (A1) and there activate three nodes—numbers 2, 3, and 4 in [Figure 6.13A](#). Activation from A1 neurons might then travel (through one or more way stations in the brain) to activate other neurons (possibly in the motor cortex) that can execute a learned behavioral response. A 560-Hz tone activates a different subset of A1 neurons—numbers 3, 4, and 5 in [Figure 6.13B](#). These subsets overlap, in that both of them contain neurons 3 and 4, as shown in [Figure 6.13C](#). Thus, an organism’s learning about the 550-Hz tone is highly likely to generalize to the 560-Hz tone. This diagram is much the same as the ones used in [Section 6.1](#) (see [Figure 6.4](#)) to show how a shared-elements representation of yellow and orange explains pigeons’ generalization between these two physically similar stimuli.

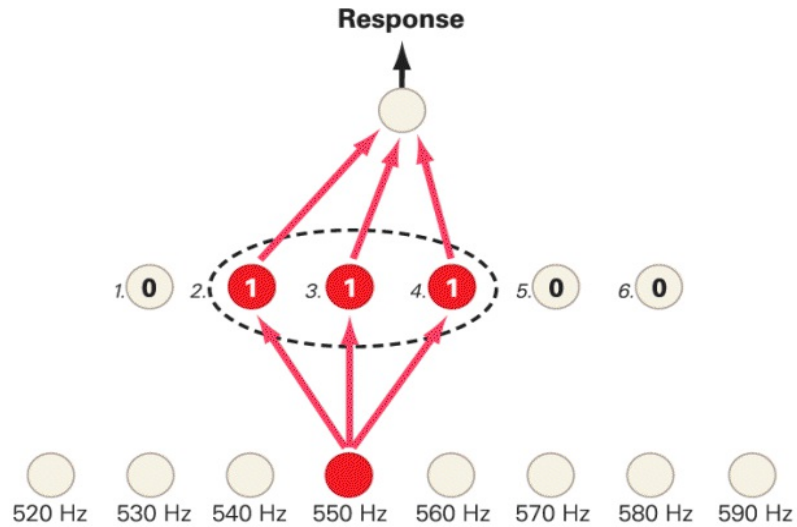


A

Output node:
motor cortex neurons
that guide a behavioral
response

Internal representation nodes:
neurons in primary
auditory cortex (A1)

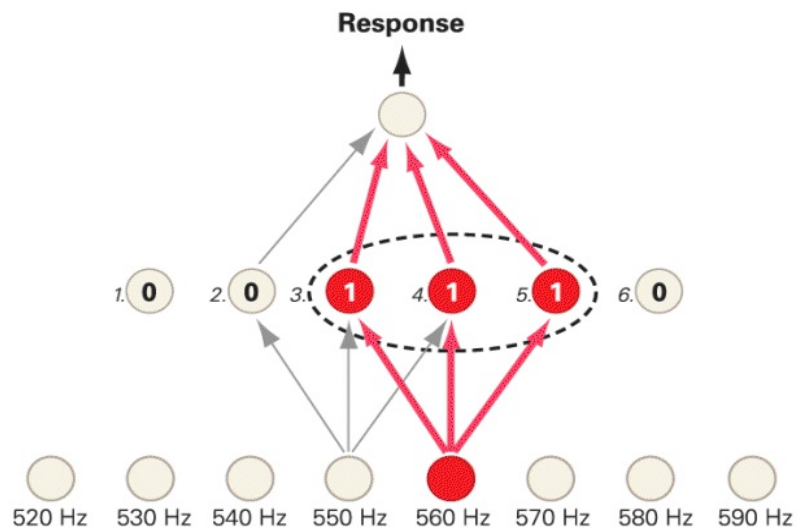
Input nodes:
sensory receptors in
the ear

**B**

Output node:
motor cortex neurons
that guide a behavioral
response

Internal representation nodes:
neurons in primary
auditory cortex (A1)

Input nodes:
sensory receptors in
the ear

**C**

Output node:
motor cortex neurons
that guide a behavioral
response

Internal representation node:
neurons in primary
auditory cortex (A1)

Input node:
sensory receptors in
the ear

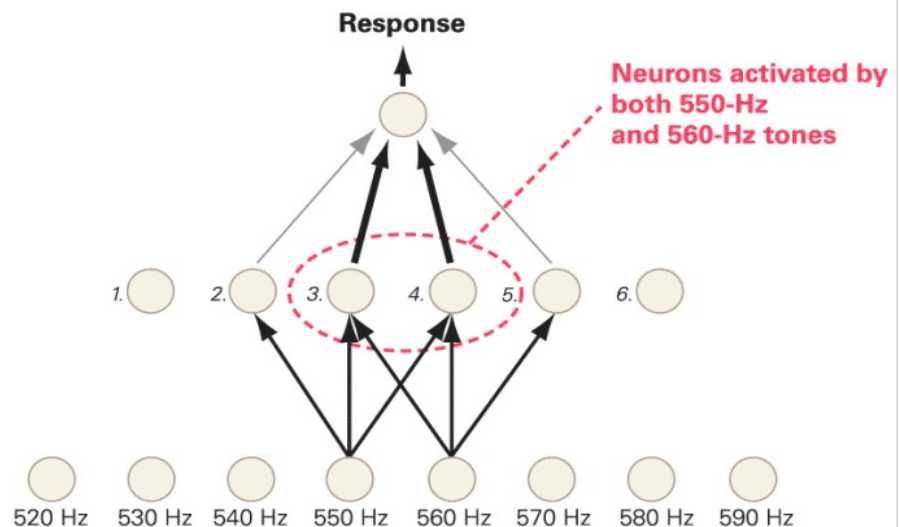
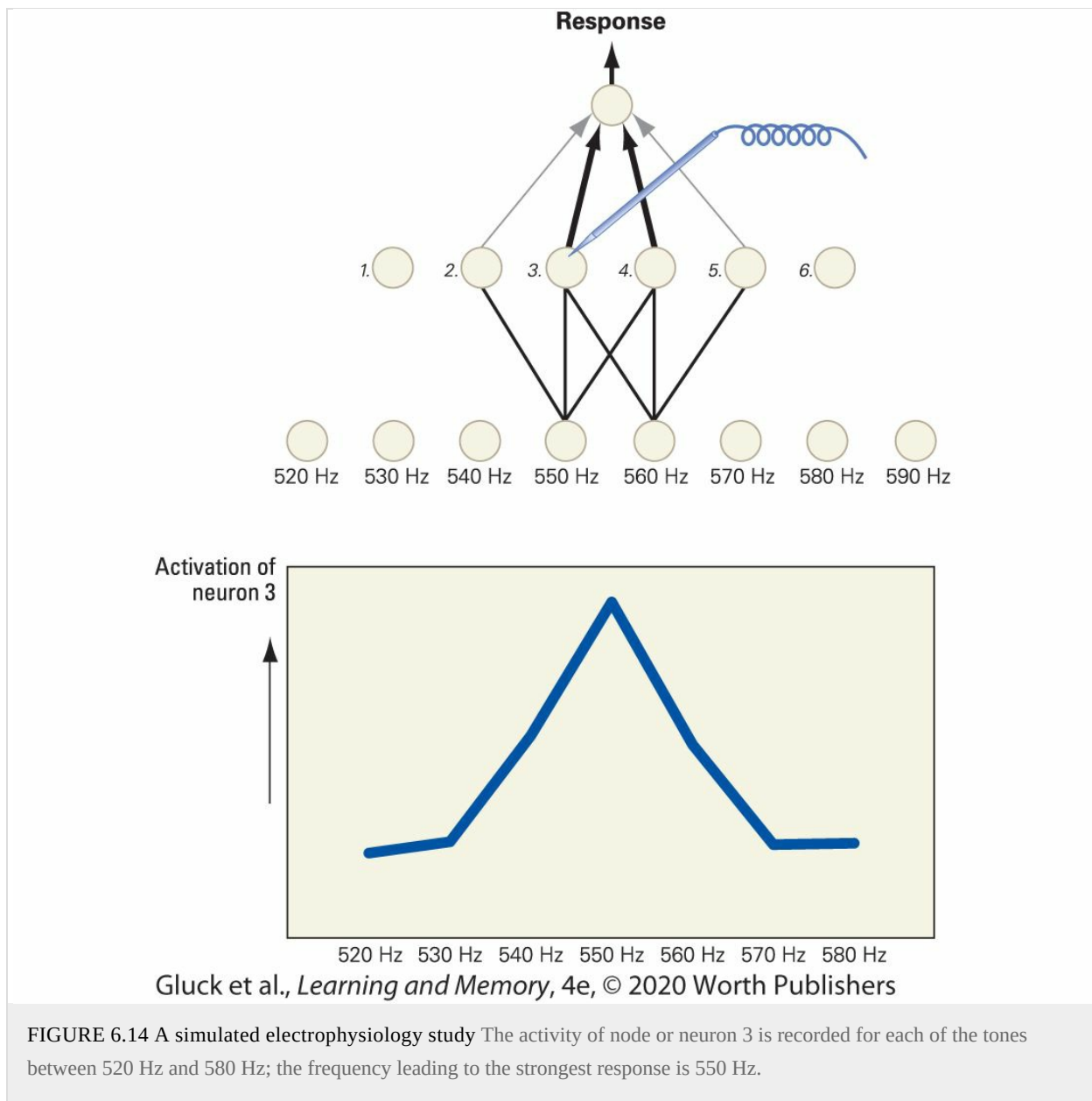


FIGURE 6.13 A shared-elements network model of generalization within the auditory cortex This network has a series of input nodes that each respond most strongly to one sound frequency and that each activate a distributed representation in the nodes of the internal representation layer. (For simplicity, only a few of the associative links are shown here.) (A) A 550-Hz tone activates nodes 2, 3, and 4 in the internal representation layer. (B) A similar, 560-Hz tone activates nodes 3, 4, and 5. (C) The overlap between these two distributed representations determines the degree to which learning about one tone will generalize to the other.

Description

All three diagrams have eight input nodes: sensory receptors in the ear, each. Each input node is represented by a circle. All the input nodes are placed side by side in a row. The input nodes from left to right are as follows. 520, 530, 540, 550, 560, 570, 580, and 590 hertz. An output node: motor cortex neuron that guides a behavioral response (represented by a circle) is above the space between the 550 and 560 hertz input node circles. Internal representation nodes: neurons in primary auditory cortex (A1) are represented by a row of circles between the input and output nodes. It has 6 circles numbered 1 to 6 from left to right, which are placed vertically above the input node circles, as follows. 530, 540, 550, 560, 570, and 580 hertz, respectively. The activation of the input nodes is represented by arrows pointing to the internal representation nodes. The activation of the internal representation nodes is represented by arrows pointing to the output node. In diagram A, input node, 550 hertz is red and activates internal representation nodes numbered 2, 3, and 4. The internal representation nodes, 2, 3, and 4, are red and numbered 1 inside each circle. All other internal representation nodes are numbered 0 inside each circle. The internal representation nodes, 2, 3, and 4, are activated. All the arrows representing activation is thick and red. The internal representation nodes, 2, 3, and 4, are encircled by dotted lines. An upward arrow from output node points to the text, response. In diagram B, input node, 560 hertz is red and activates internal representation nodes numbered 3, 4, and 5. The internal representation nodes, 3, 4, and 5, are red and numbered 1 inside each circle. All other internal representation nodes are numbered 0 inside each circle. The activation of the 550 and 560-hertz input node is represented by thin gray and thick red arrows, respectively. The activation of internal representation nodes, 3, 4, and 5, are represented by thick, red arrows. The activation of internal representation node 2 is represented by a thin, gray arrow. The internal representation nodes, 3, 4, and 5, are encircled by dotted lines. An upward arrow from output node points to the text, response. In diagram C, input node, 550 hertz activates internal representation nodes numbered 2, 3, and 4. The input node, 560 hertz activates internal representation nodes numbered 3, 4, and 5. The activation by the 550 and 560-hertz input nodes is represented by thin, black arrows. The activation of internal representation nodes, 3 and 4, are represented by thick, black arrows. The activation of internal representation nodes 2 and 5 are represented by thin, gray arrows. The internal representation nodes, 3 and 4, are encircled by dotted lines and labeled neurons activated by both 550-hertz and 560-hertz tones. An upward arrow from output node points to the text, response.

The simplified network in [Figure 6.13](#) may explain why cortical neurons display receptive fields. For each tone in a continuum, you can ask how a particular A1 neuron, such as neuron 3, will respond. The curve in [Figure 6.14](#) shows the results we would expect. The neuron's best frequency is 550 Hz; similar tones also activate this neuron, although not as strongly as a tone of 550 Hz. The result is a generalization gradient that looks very like the actual receptive fields observed during cortical mapping studies, like the one shown in [Figure 3.12](#).



Description

The diagram has eight input nodes. Each input node is represented by a circle. All the input nodes are placed side by side in a row. The input nodes from left to right are as follows. 520, 530, 540, 550, 560, 570, 580, and 590 hertz. An output node (represented by a circle) is above the space between the 550 and 560-hertz input node circles. Internal representation nodes are represented by a row of circles between the input and output nodes. It has 6 circles numbered 1 to 6 from left to right, which are placed vertically above the input node circles, as follows. 530, 540, 550, 560, 570, and 580 hertz, respectively. Input node 550 hertz activates internal representation nodes numbered, 2, 3, and 4, which in turn activates the output node. Input node 550 hertz activates internal representation nodes numbered, 3, 4, and 5, which in turn activates the output node. The activation of internal representation nodes by the input nodes are represented by lines. The activation of the output node by the internal representation nodes are represented by arrows. The arrows from the internal representation nodes numbered, 2 and 5, are thin, gray. The arrows from the internal

representation nodes numbered, 3 and 4, are thick, black . An electrode is placed on the internal representation node numbered 3. An accompanying graph has a vertical axis labeled, activation of neuron 3. The horizontal axis ranges from 520 to 580 hertz, in increments of 10 hertz. A line starts above 520 hertz in the horizontal axis, increases to a maximum point above 550 hertz. The increase between 540 and 550 hertz is the steepest, followed by the increase of the line between 530 and 540 hertz. From the maximum point, the line decreases to end at a point above 580 hertz. The decrease between 550 and 560 hertz is the steepest, followed by a decrease between 560 and 570 hertz. The increase and the decrease of the line are symmetrical about 550 hertz.

Evidence of Primary Cortex Role in Generalization

The idea of topographic organization and brain regions dedicated to specific tasks was a central part of Pavlov's theories of learning in the early 1920s, but it remained only a theoretical conjecture until nearly a half century later. In the 1960s, Richard Thompson established a direct relationship between behavioral properties of auditory generalization and certain anatomical and physical properties of the auditory cortex ([Thompson, 1962](#)).

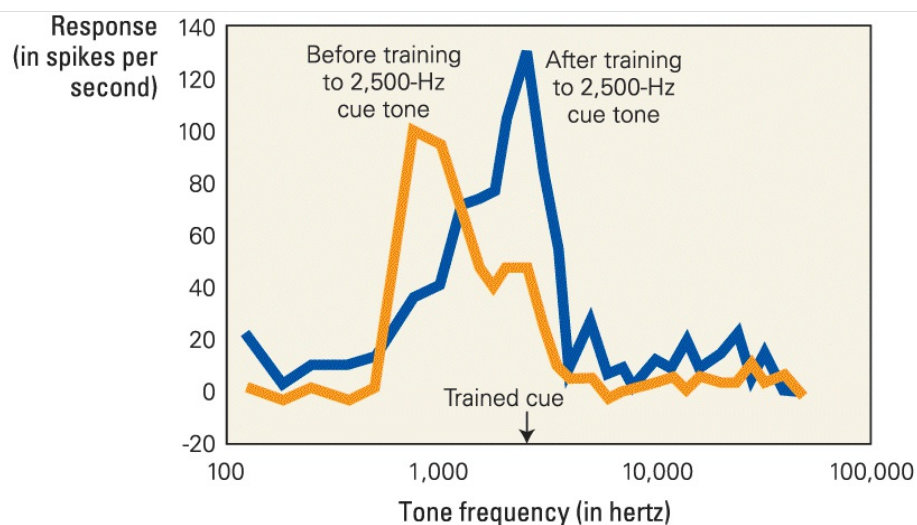
A common experimental finding was that cats trained to respond to a tone of a particular frequency would show a generalization gradient to tones of other frequencies, much like the sloping gradient seen in [Figure 6.1](#). However, after Thompson removed the primary auditory cortices (A1) of some of the cats, they responded equivalently to all tones, even those separated by five octaves or more! This experiment demonstrated that A1 was necessary for the production of appropriate generalization gradients to auditory stimuli ([Thompson, 1965](#)). The indiscriminate behavior of the lesioned cats reflected massive overgeneralization, resulting in a flat generalization gradient. As a control study, Thompson also tested cats who had undergone removal of the primary somatosensory cortex (area S1) instead. These animals showed normal generalization behavior with regard to tones, indicating that the auditory overgeneralization occurred specifically in animals with A1 lesions. Similar overgeneralization of visual stimuli has been reported in monkeys with damage to V1, the primary visual cortex ([Thompson, 1965](#)).

These studies suggest that, although it is possible for an animal to learn to respond to stimuli while lacking the corresponding areas of sensory cortex, an intact sensory cortex for that stimulus type is essential for normal generalization. Thus, without A1, animals can learn to respond to the presence of a tone but cannot respond precisely to a *specific* tone. In other words, without the primary sensory cortex, animals overgeneralize and have difficulty discriminating stimuli in the corresponding sensory modality. What these early studies did *not* show is whether

sets of neurons in the brain can learn new responses as a result of experience. Later studies showed that they can.

Plasticity of Cortical Representations

In seminal studies on the neural bases of learning and cortical plasticity, Norman Weinberger and his colleagues recorded responses from individual neurons in the primary auditory cortices of guinea pigs before and after the animals were trained to respond to auditory cues ([Weinberger, 1993](#)). In one study, Weinberger and colleagues recorded the activity of neurons in A1 before and after the animals experienced presentations of a 2,500-Hz tone paired with shock. Through training, many neurons changed their receptive field to become most responsive to tones near the training frequency of 2,500 Hz. One such neuron, shown in [Figure 6.15](#), had originally responded most strongly to tones of about 1,000 Hz but now responded most strongly to tones of the trained frequency. These cortical changes occurred quickly, after as few as five pairings of the tone and shock. If enough neurons were to show this type of change, the overall result could amount to cortical remapping that allows a larger area of A1 to respond to the trained frequency.



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

FIGURE 6.15 Plasticity of representation in the primary auditory cortex After training in which a 2,500-Hz tone predicted a shock, the response of an A1 neuron changed from previously being most responsive to a 1,000-Hz tone to being most responsive to tones nearer to the training frequency.

Data from [Weinberger, 1977](#), Figure 2.

Description

The horizontal axis is labeled tone frequency (in hertz), with 100, 1,000, 10,000, and 100,000 marked on it. The line for

before training to 2,500-hertz cue tone starts at (200, 0), continues with fluctuations until (800, 0). It then increases to a maximum at (900, 100). It then decreases to (9,000, 0) and continues along 0 responses with fluctuations, before ending at (90,000, 0). The line for after training to 2,500-hertz cue tone starts at (200, 20), decreases to (300, 5). It then increases to a maximum at (2,500, 100). It then decreases to (6,000, 0) and continues between 0 and 20 responses with fluctuations, before ending at (90,000, 0).

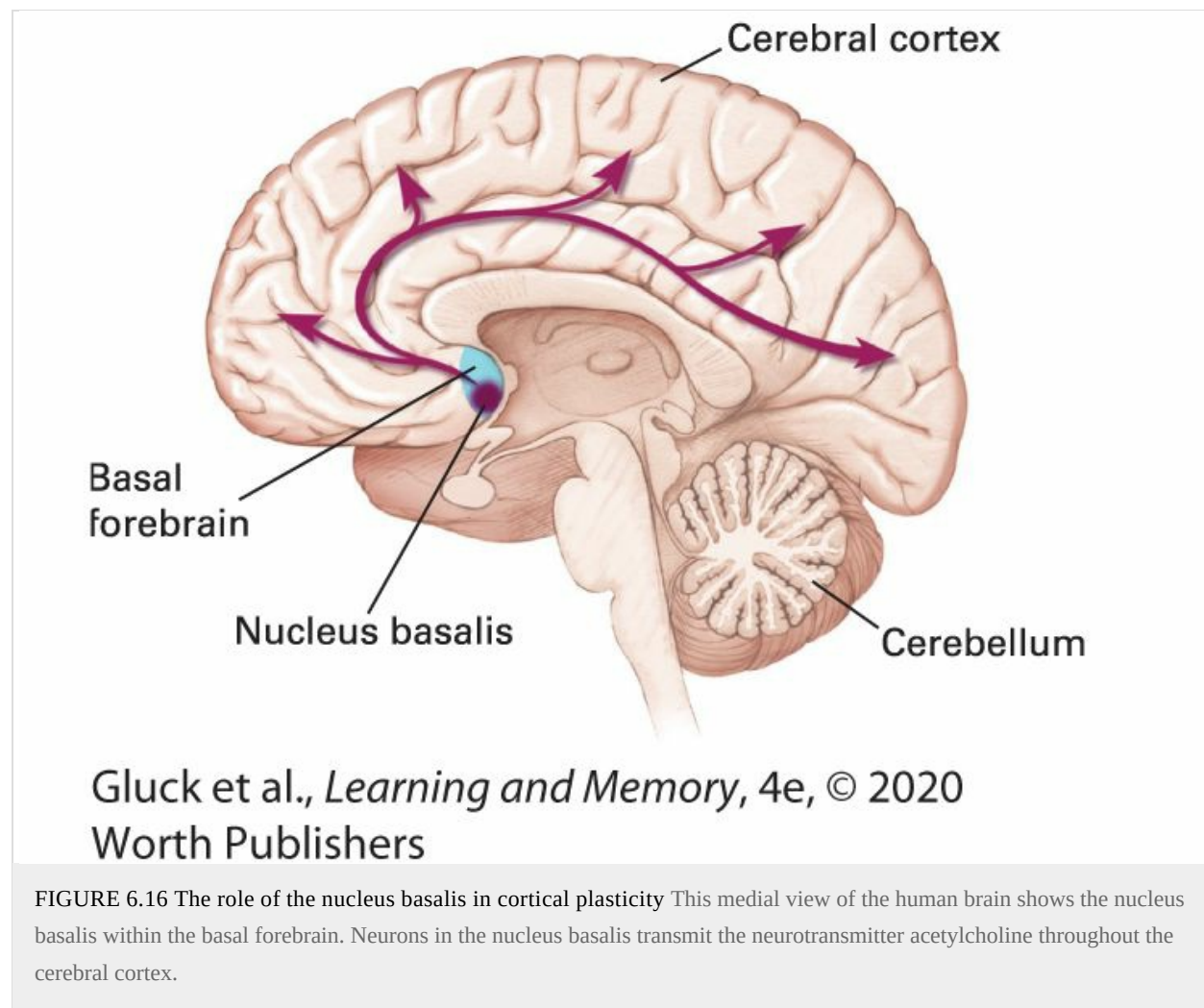
In other studies, Weinberger showed that if a tone is repeatedly presented alone (as in habituation, described in [Chapter 3](#)), the opposite effect occurs: there is a decrease in neuronal responding to this frequency ([Condon & Weinberger, 1991](#)). Moreover, if the tone and shock are both presented but not paired (i.e., if they are presented separately), then no significant changes are observed in the neurons' responses to tones ([Bakin & Weinberger, 1990](#)). This result indicates that the cortical plasticity is a result of the tone–shock pairing. It implies that stimulus presentation alone doesn't drive cortical plasticity; the stimulus has to be meaningfully related to ensuing consequences, such as a shock.

If cortical change occurs because a stimulus in one sensory modality is meaningfully related to—or, in other words, is predictive of—a salient consequence, such as food or shock, in a different sensory modality, how did information about that consequence reach the primary sensory cortex of the first modality in order to produce the change there? After all, A1 is specialized to process information about sounds, while food is a gustatory (taste) stimulus, and shocks are somatosensory, not auditory, stimuli. Yet the findings in [Figure 6.15](#) clearly indicate that pairing a tone with a shock *does* bring about a change in the auditory cortex.

Weinberger has argued that A1 does not receive specific information about somatosensory or gustatory stimuli but rather receives only information that some sort of salient event has occurred ([Weinberger, 2004](#)). This information is enough to instigate cortical remapping and expand the representation of the cue stimulus. The primary sensory cortices (A1, V1, S1, and so on) only determine which stimuli deserve expanded representation within that primary cortex and which do not.

How does the brain determine whether a stimulus merits cortical remapping, without necessarily specifying exactly why? It turns out that several brain regions may serve this function. The basal forebrain is a group of nuclei important for learning and memory; damage to it can produce *anterograde amnesia*, which is a severe impairment in forming new fact and event memories

(more on this in [Chapter 7](#)). Many cortical mapping researchers have focused on a small group of neurons located in an area of the basal forebrain called the **nucleus basalis** ([Figure 6.16](#)), which projects to all areas of the cortex and to the amygdala. When nucleus basalis neurons are activated, they release **acetylcholine (ACh)**, a neurotransmitter that has many functions in the brain, including the promotion of neuronal plasticity. In summary, the nucleus basalis functions to enable cortical plasticity: when a CS is paired with a US, the nucleus basalis becomes active and delivers acetylcholine to the cortex, enabling cortical remapping to enlarge the representation of that CS ([Weinberger, 2003](#)).



Description

Arrows from the nucleus basalis project toward the frontal portion of the brain. These arrows then bend to the back of the brain above the cerebellum in the cerebral cortex.

nucleus basalis

A small group of neurons located in the basal forebrain; these neurons deliver acetylcholine to the cortex, enabling cortical plasticity.

acetylcholine (ACh)

A neuromodulator that strongly influences hippocampal function.

But how does the nucleus basalis “know” when to become active? It receives the information through connections from areas such as the *amygdala*, which codes emotional information such as discomfort and pain (e.g., from an electric shock) and pleasure (e.g., from food). (The function of the amygdala is discussed further in [Chapter 10](#).)

Several studies have confirmed that the nucleus basalis can play a role in mediating cortical plasticity. Most importantly, experiments show that if a tone is paired with nucleus basalis stimulation—rather than with a “real” consequence, such as food or shock—cortical remapping occurs to enhance response to that tone ([Bakin & Weinberger, 1990](#); [Kilgard & Merzenich, 1998](#)). These findings are exciting because of their implications for rehabilitation after cortical damage. It may eventually be possible to use judicious stimulation of the nucleus basalis to encourage cortical remapping in individuals who have lost the use of one of their cortical areas.

TEST YOUR KNOWLEDGE

Cholinergic Neurons and Learning

In an experiment to test the function of specific cholinergic (acetylcholine-releasing) neurons in learning and memory, Jackie makes a cut through cholinergic neurons in the nucleus basalis of the rat brain. How will this lesion affect discrimination learning the rats acquired before the surgery? How will it affect discrimination learning after the surgery? ([Answers appear at the end of the chapter.](#))

Generalization and the Hippocampal Region

Many forms of learning require sensitivity to the relationship between multiple sensory cues; two examples are the sensory preconditioning and acquired-equivalence processes discussed in [Section 6.1](#). Learning about relationships between stimuli in the environment is part of the special role the hippocampus and related structures serve in learning and conditioning. This portion of the brain is most often referred to as the [hippocampal region](#) in rats and other small animals and as the [medial temporal lobe](#) in humans ([Figure 6.17](#); see also [Figure 4.21](#)).

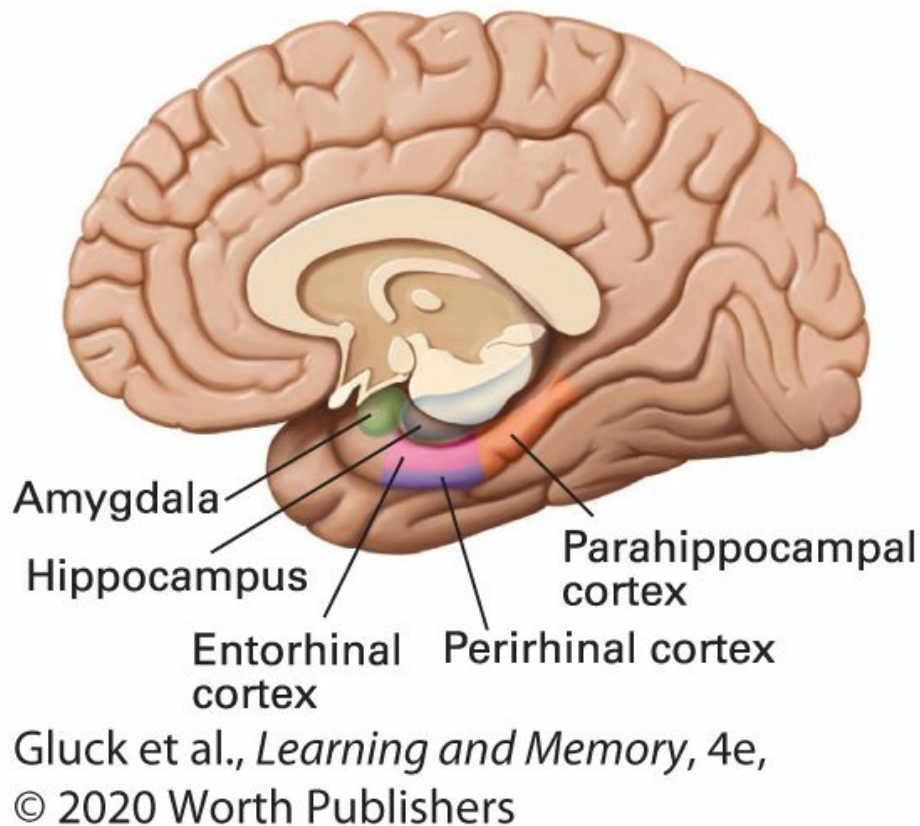


FIGURE 6.17 The hippocampus and medial temporal lobe in humans

hippocampal region

The hippocampus and associated brain regions, including the entorhinal cortex and dentate gyrus. In humans, also referred to as the *medial temporal lobe*.

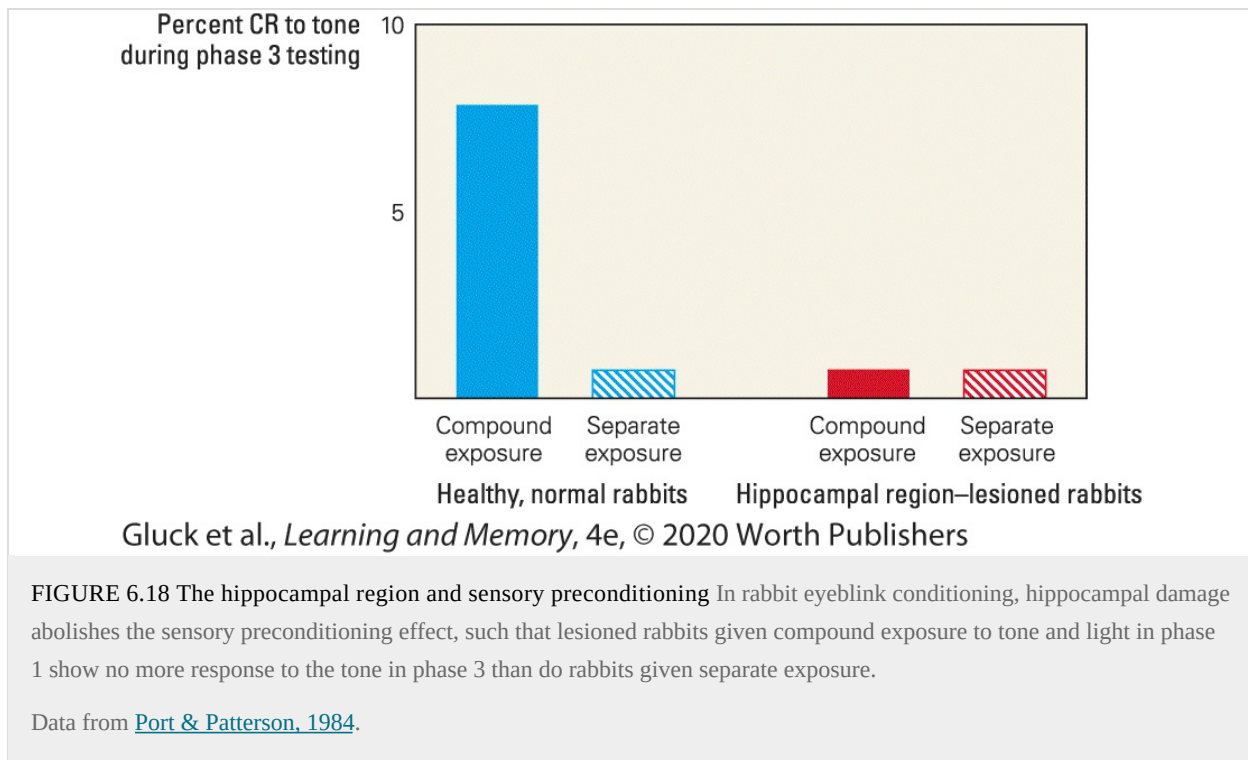
medial temporal lobe

The medial (or inner) surface of the temporal lobe that contains the hippocampus, the amygdala, and other structures important for memory.

Effect of Damage to the Hippocampal Region

To examine the role of the hippocampal region in generalization, we must return to the procedure for sensory preconditioning, summarized in [Table 6.2](#). In the first phase of sensory preconditioning experiments, animals in the compound exposure group are exposed to a combination of two stimuli, such as a tone and a light, presented together. Control group animals are exposed to each stimulus separately. In phase 2, presentation of one of the stimuli by itself—say, the light—predicts a salient event, such as a blink-evoking airpuff; control animals and compound exposure animals learn to respond to the light with an eyeblink. Finally, in phase 3, the two groups of animals are tested with the other stimulus (in this case, the tone) alone. Normal

animals of the compound exposure group will give a significant eyeblink response to the tone in phase 3, indicating that they have generalized the training from the light to the tone based on the prior co-occurrence of tone and light ([Figure 6.18](#)). By contrast, normal animals of the control group, given separate exposure to the tone and light in phase 1, show little or no responding to the tone in phase 3.



Description

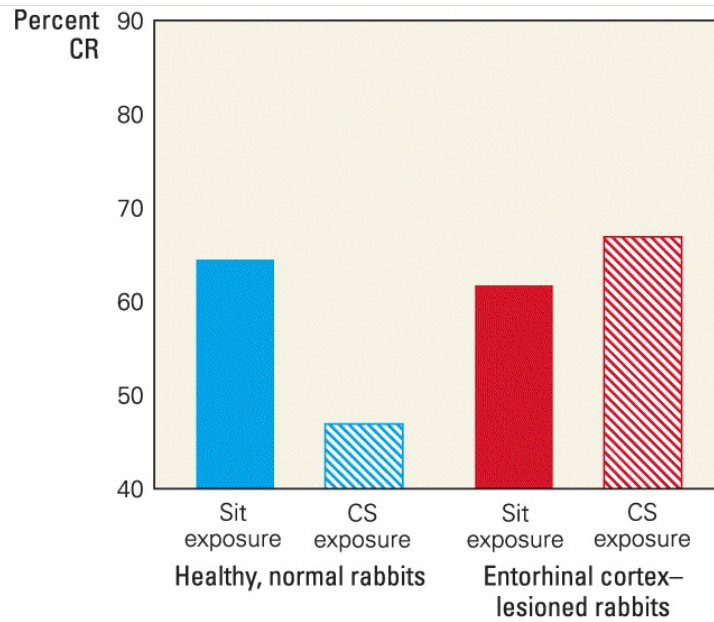
The vertical axis represents percent C R to tone during phase 3 testing, ranging from 0 to 10, in increments of 5. The percent C R to tone of healthy normal rabbits during compound exposure is 8 and during separate exposure is 1. The percent C R to the tone of hippocampal region lesioned rabbits during compound exposure is 1 and during separate exposure is 1. All values are estimated.

The results are different, however, for animals with damage to their hippocampal region. Rabbits with surgically created lesions in the fornix (a part of the hippocampal region) display no sensory preconditioning ([Figure 6.18](#); [Port & Patterson, 1984](#)). That is, lesioned animals in the compound exposure group show no more transfer in phase 3 than do animals in the separate exposure group. It seems that an intact and functioning hippocampal region is needed for generalizing between stimuli that have co-occurred in the past.

Similarly, in a study of acquired equivalence using rats (see [Table 6.3](#)), researchers found that rats with hippocampal-region damage—specifically, lesions of the entorhinal cortex, which lies next to the hippocampus—show impaired acquired equivalence: they can learn the associations in phases 1 and 2 but show no generalization in phase 3 ([Coutureau et al., 2002](#)). Here again, animal lesion studies show that the hippocampal region is critical for generalization.

Other evidence supporting a key role for the hippocampal region in generalization comes from studies of latent inhibition, a classical conditioning paradigm in which learning a CS–US association is slower in animals that are given prior exposure to the CS (*CS exposure* condition) compared with animals that are given equivalent prior exposure to the context alone (*sit exposure* condition; [Table 4.7](#) and [Figure 4.15](#)). This is essentially a generalization task in which the CS-exposed animals are tricked into overgeneralizing from phase 1 (where there is no US) to phase 2 (where the CS now does predict a US).

One such finding is shown in [Figure 6.19](#), from a study demonstrating that latent inhibition in rabbit eyeblink conditioning is eliminated by hippocampal-region damage—specifically, a lesion of the entorhinal cortex ([Shohamy, Allen, & Gluck, 2000](#)). One interpretation of latent inhibition is that the experimental procedure manipulates the relationship between the stimulus cue and the context during pre-exposure. According to this view, in the CS-exposed group, the CS is generalized to the context during phase 1 and therefore is harder to discriminate from the context in phase 2, when the animal encounters the CS in the same context and must learn that it now predicts a US. Consistent with this idea, other studies have shown that latent inhibition is highly context sensitive and requires that the animal be exposed (phase 1) and trained (phase 2) in the same context ([Lubow, 1989](#)). This may explain why people who have sustained damage to their hippocampal region fail to show latent inhibition despite being able to show normal acquisition of a learned response ([Myers et al., 2000, 2003b](#)).



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FIGURE 6.19 Latent inhibition in rabbit eyeblink conditioning Latent inhibition in rabbit eyeblink conditioning is eliminated by hippocampal-region damage, specifically a lesion of the entorhinal cortex. When control rabbits did not receive any pre-exposure to the tones (“Sit exposure” condition, solid green bar), they produced many more eyeblink CRs during subsequent tone–airpuff training than did control rabbits that had been previously exposed to the tone with no airpuff US (green-striped bar). In contrast, animals with entorhinal cortex (EC) lesions (shown in red) showed no such effect. In fact, the rabbits with EC lesions in the “CS exposure” group (red-striped bar) learned better than control rabbits in the equivalent CS exposure group (green-striped bar). Brain damage actually helped these rabbits learn faster.

Data from [Shohamy, Allen, & Gluck, 2000](#).

Description

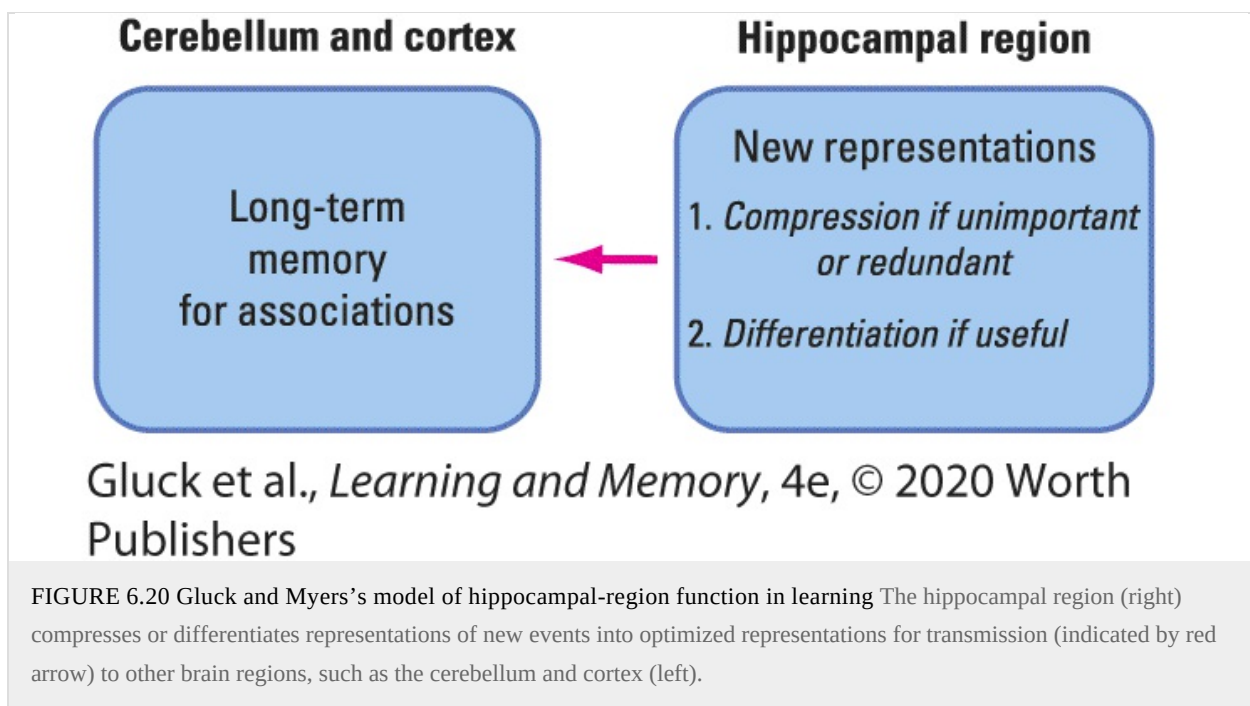
The vertical axis represents percent C R, ranging from 40 to 90, in increments of 10. The percent C R of healthy normal rabbits during sit exposure is 64, and during C S exposure is 46. The percent C R of entorhinal cortex lesioned rabbits during sit exposure is 62 and during separate exposure is 67. All values are estimated.

In summary, results from studies of hippocampal-region lesions in animals, using three different experimental paradigms—sensory preconditioning, acquired equivalence, and latent inhibition—all converge to suggest that this brain region is critical for stimulus generalization, especially when that generalization involves learning relationships between different stimuli.

Modeling the Role of the Hippocampus in Adaptive Representation

The data described previously suggest that the hippocampal region is involved in even the most elementary forms of associative learning, including classical conditioning. ([Chapter 7](#) shows how the hippocampus and related structures in the medial temporal lobe are critical for learning about facts and events, too.) This does not mean that the hippocampal region is necessary for learning a stimulus–response association. Rather, the hippocampal region appears to be critically involved in how newly acquired associations are *represented*.

Mark Gluck and Catherine Myers proposed a model in which the hippocampal region operates as an “information gateway” during associative learning, processing new representations of events that are experienced ([Gluck & Myers, 1993, 2001](#)). In their model, illustrated in [Figure 6.20](#), the hippocampal region selects what information is allowed to enter memory and how it is to be encoded by other brain regions. Specifically, Gluck and Myers proposed that the representation of unimportant or redundant information undergoes shrinkage (or compression) by the hippocampal region, whereas the representation of useful information is expanded (or differentiated), creating a new, efficient, optimized representation that encodes only the key aspects of incoming information. They describe the modification process as “adaptive representation.”



Description

Hippocampal region has new representations: 1 compression if unimportant or redundant and 2 differentiation if useful.
The cerebellum and cortex have long-term memory for associations.

Recall [Figure 6.11](#), which caricatures a typical New Yorker's mental map of the world. This image captures just the kind of compression (e.g., of the entire Midwest into a small, barren strip) and expansion (e.g., the exaggerated detail of Ninth and Tenth Avenues) that Gluck and Myers propose are dependent on the hippocampal region. In their model, these representational changes are computed in the hippocampal region and then used by other brain regions, such as the cerebral cortex and the cerebellum, where the stimulus–response associations that control motor outputs are actually stored.

Gluck and Myers applied their model of the hippocampal region's role in conditioning to a broad range of experimental findings, including the studies of sensory preconditioning and latent inhibition summarized earlier. In all cases, they showed that the learning displayed by healthy, normal animals was similar to how the model behaved when the representational compression and differentiation processes were turned on. In contrast, when these hippocampal-dependent changes in representation were turned off, the resulting “lesioned” model provided a good description of the altered learning seen in animals with lesions to their hippocampal region, as in the studies of sensory preconditioning, acquired equivalence, and latent inhibition ([Gluck & Myers, 2001](#)).

Although brain damage in animals and humans is usually not considered desirable, there are select situations in which brain damage, specifically hippocampal-region damage, can actually facilitate learning and performance. These situations are predicted by the [Gluck and Myers \(1993\)](#) model to be those in which the absence of adaptive representation during learning (in other words, where the representations stay fixed and rigid) enhances performance in a subsequent task. In one confirmation of this projection, the model predicted correctly that patients with hippocampal-region damage and global amnesia who are trained to learn about high-distortion stimuli (such as those in [Figure 6.6B](#)) would later generalize more accurately to novel high-distortion items ([O'Connell et al., 2016](#)).

Further evidence implicating hippocampal-region function in the modification of stimulus representations in human learning and generalization comes from functional brain imaging. The

Gluck and Myers model predicts that the hippocampal region should be very active early in training, when subjects are learning about stimulus–stimulus regularities and developing new stimulus representations, but that it should be less active later in training, when other brain regions (such as the cerebellum and cerebral cortex) are using these representations to perform the behavioral response. A functional magnetic resonance imaging (fMRI) study of normal humans learning a probabilistic categorization task confirmed the prediction that activity in the hippocampal region was high early in training and then tapered off as the task was learned ([Poldrack et al., 2001](#)).

Interim Summary

- Although animals can learn to respond to auditory stimuli without the primary auditory cortex, an intact A1 is essential for normal auditory learning and generalization. Without A1, animals can learn to respond to the presence of a tone but cannot respond precisely to a specific tone.
- Cortical plasticity is driven by the correlations between stimuli and salient events, with the nucleus basalis mediating between the amygdala (which decides what stimuli are salient) and the cortex.
- The hippocampal region plays a key role in forms of learning that depend on stimulus generalization, including the classical conditioning paradigms of acquired equivalence, sensory preconditioning, and latent inhibition.
- Modeling suggests that one role of the hippocampal region is to bring about compression or differentiation of stimulus representations, as appropriate.

6.3 Clinical Perspectives

In the final section of the chapter, we consider some of the consequences when generalization breaks down and leads to incorrect predictions about the world around us. We focus here on two such disorders: schizophrenia and autism.

Generalization Deficits in Schizophrenia

Given what animal studies have shown about the importance of the hippocampal region in generalization, it will not surprise you to learn that damage or dysfunction in the hippocampal region in humans can also have deleterious consequences for learning and generalization. For example, hippocampal dysfunction is observed in the mental disorder schizophrenia, a complex and heterogeneous disorder that involves many brain areas. The dysfunction of the hippocampus in these patients leads to significant changes in how they learn and generalize, impairing their interaction with the world, especially in novel situations. ([Chapter 9](#) addresses some of the ways in which frontal lobe dysfunction impairs aspects of learning, memory, and attention in schizophrenia.)

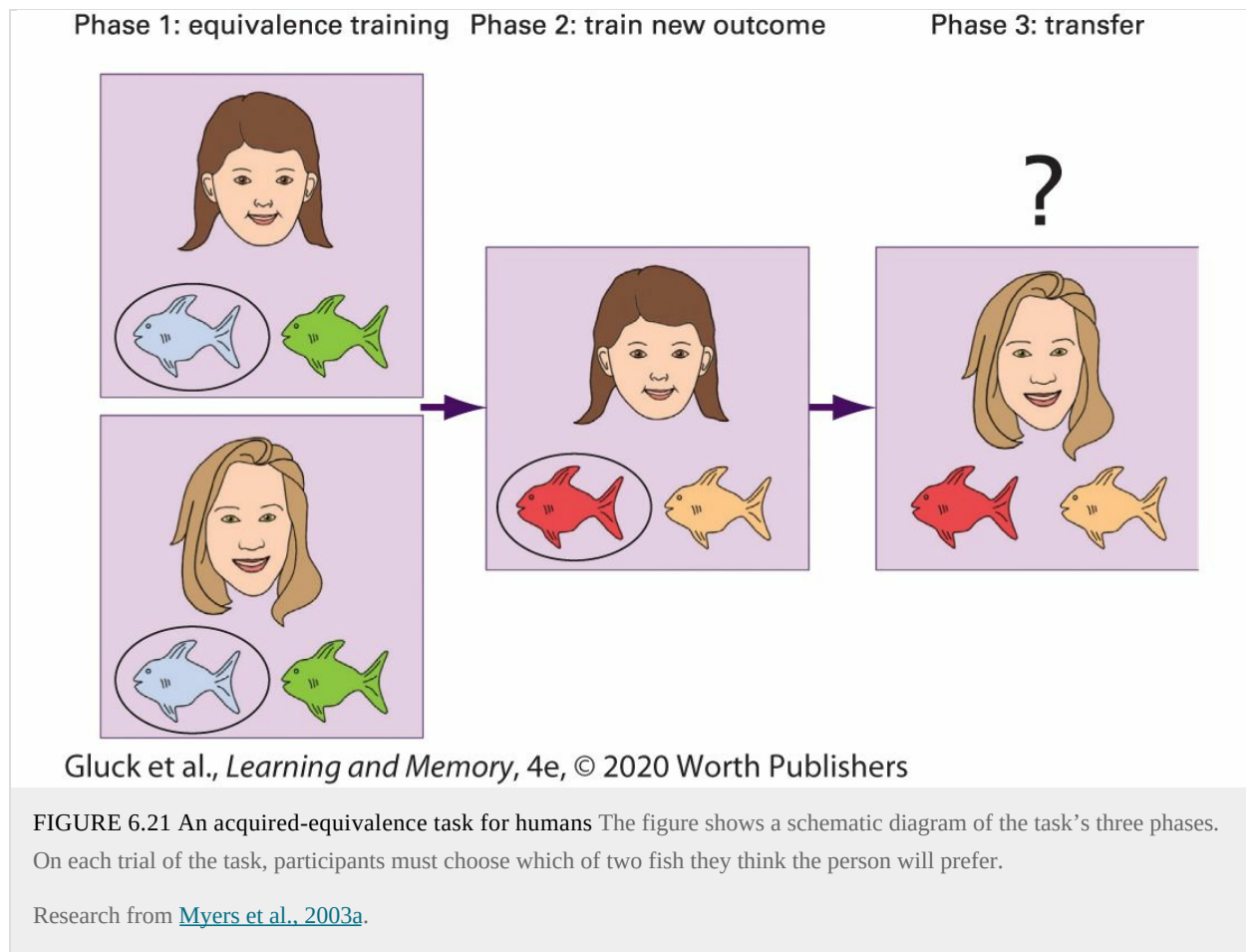
Schizophrenia is a severe mental disorder with symptoms of hallucinations, delusions, flattened affect, and social impairment. Functional brain-imaging studies of patients diagnosed with schizophrenia have shown reduced hippocampal activity, and a wide range of structural and functional brain-imaging studies have identified abnormalities in the hippocampus as a core feature of the illness, present from the onset and, to a lesser degree, also seen in first-degree relatives of people diagnosed with schizophrenia ([Heckers, 2001](#); [Heckers et al., 1998](#)). In particular, people diagnosed with schizophrenia show abnormalities in their hippocampal shape, most notably an overall smaller volume.

More recently, studies of conditioning and generalization have shown that most patients diagnosed with schizophrenia are able to learn simple associations but are markedly impaired when these associations must be transferred to a new context ([Polgár et al., 2008](#)) or flexibly modified in some other way ([Waltz & Gold, 2007](#)). Studies like the ones discussed next are helping us understand how some of the cognitive impairments in schizophrenia may be related to dysfunction in the hippocampus.

Acquired Equivalence in Schizophrenia

Catherine Myers and colleagues have adapted the acquired-equivalence procedure from animal conditioning for use with humans ([Myers et al., 2003a](#)). In consideration of the data that the hippocampal region in rats is essential for acquired equivalence ([Coutureau et al., 2002](#)), Myers and colleagues applied their human version of the task to the study of generalization impairments in people with schizophrenia.

On each of several trials in the Myers et al. human acquired-equivalence tasks, participants see a cartoon face and two colored fish and are asked to learn which fish each person prefers, as shown in [Figure 6.21](#). In phase 1, for example, the participants might be asked to learn that a certain brown-haired girl prefers blue fish over green fish and that a certain other person (a blond-haired woman) also prefers blue fish over green fish ([Figure 6.21](#), left). Participants should gradually learn that these two people are equivalent in the sense that they are to be paired with the same fish. In phase 2, participants learn some new information: the brown-haired girl also prefers red fish over yellow fish ([Figure 6.21](#), center). Finally, phase 3 is a testing phase, in which participants are quizzed on all the pairings they have learned so far. Intermixed with this testing are some critical pairings that the participants have never seen before. For example, they are shown the blond-haired woman and asked if she is likely to prefer a red or a yellow fish ([Figure 6.21](#), right).



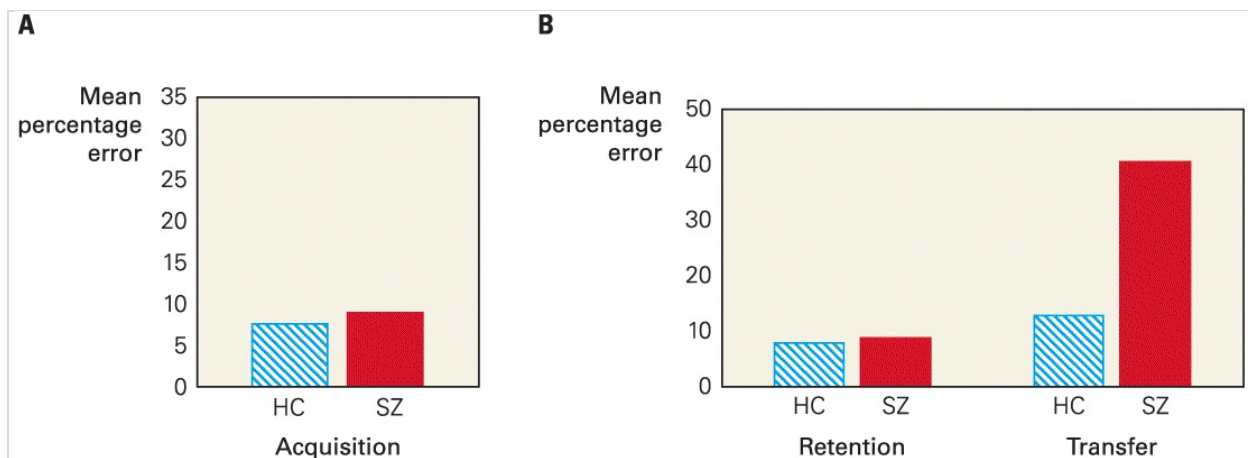
Description

Phase 1 is on the left, phase 2 at the center, and phase 3 on the right. Each phase leads to the next phase and is connected by a rightward arrow. In phase 1, equivalence training, both the brown-haired woman and the blonde woman prefer a blue fish over a green fish. In phase 2, train new outcome, the brown-haired woman prefers a red fish over a yellow fish. In phase 3, transfer, the blonde woman is presented with a red and a yellow fish. A question mark is above the blonde woman.

Healthy adults reliably judge that the blond-haired woman prefers red fish over yellow fish, even though they have never specifically been taught this ([Myers et al., 2003a](#)). In essence, they are showing acquired equivalence: when they learn that two people show an equivalent pattern of past preferences (as in phase 1) and that one of the two prefers the red fish over the yellow fish (in phase 2), they judge that the other person will probably prefer the red fish over the yellow fish as well.

How do people with schizophrenia perform on this task? Szabolcs Kéri and colleagues in

Hungary have conducted several studies using the task from [Figure 6.21](#) to compare patients with schizophrenia and control subjects ([Farkas et al., 2008](#); [Kéri, Nagy, Kelemen, Myers, & Gluck, 2005](#)). The deficit observed in the generalization phase in patients with schizophrenia suggests that hippocampal-region–dependent functions are indeed impaired in schizophrenia ([Figure 6.22](#)). More recently, antipsychotic medications have been shown to partially remediate the acquired-equivalence deficits in people diagnosed with schizophrenia, suggesting that these medications either enhance hippocampal-region function directly or else indirectly enhance the ability of the hippocampal region to cooperate with other brain regions ([Shohamy et al., 2010](#)).



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FIGURE 6.22 Acquired-equivalence data from people diagnosed with schizophrenia When tested on the acquired-equivalence task from [Figure 6.21](#), patients diagnosed with schizophrenia (SZ) are normal at learning and retention of associations but are impaired at transfer generalization as compared with matched healthy controls (HC).

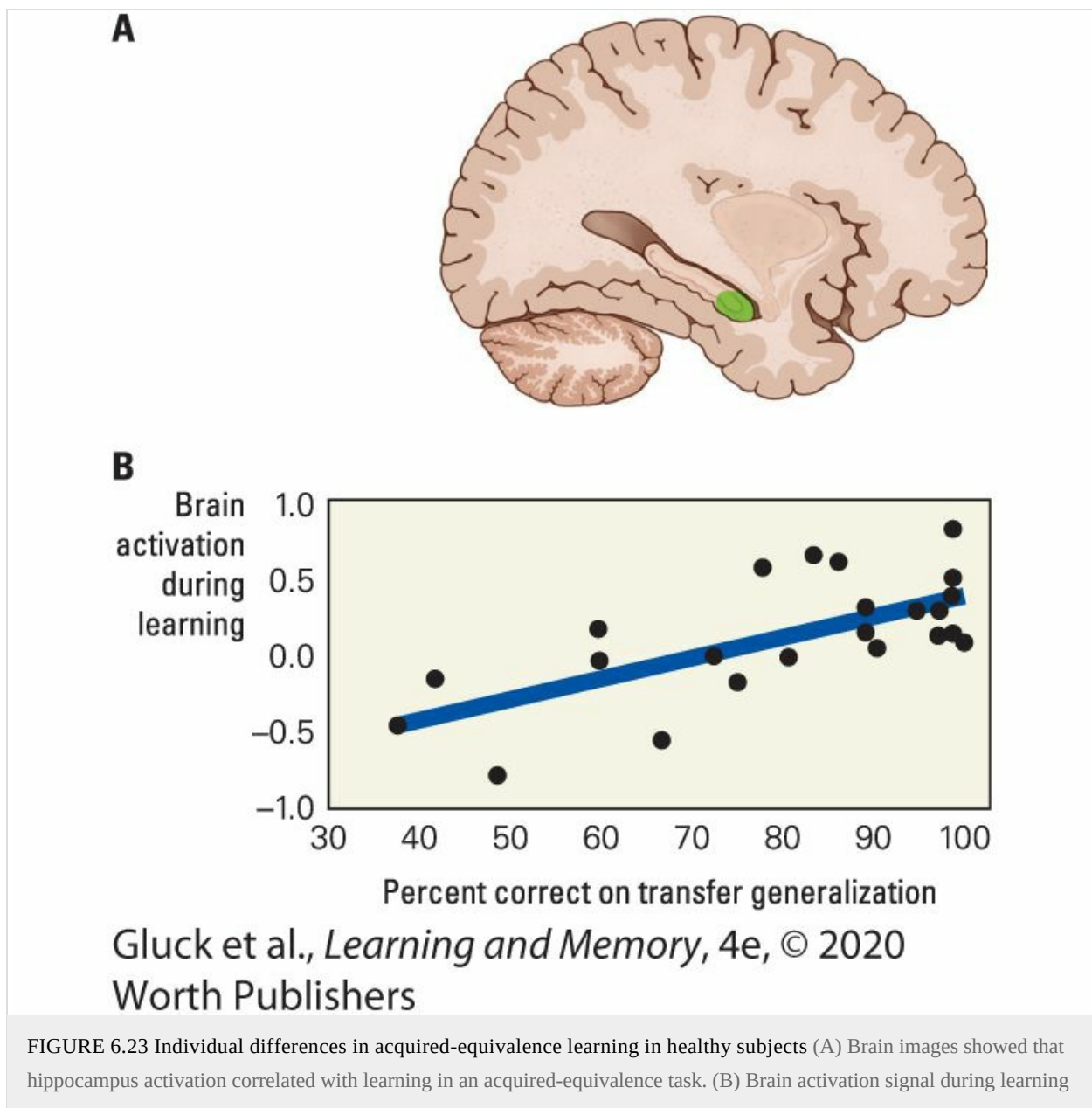
Data from [Kéri et al., 2005](#).

Description

Graph A compares the mean percentage error of H C and S Z accusation. The vertical axis represents mean percentage error, ranging from 0 to 35, in increments of 5. For acquisition, the mean percentage error in H C is 7, and in S Z is 9. Graph B compares the mean percentage error of H C and S Z during retention and transfer. The vertical axis represents mean percentage error, ranging from 0 to 60, in increments of 10. For retention, the mean percentage error in H C is 8, and in S Z is 9. For transfer, the mean percentage error in H C is 12, and in S Z is 42. All values are estimated.

To further clarify the role of the hippocampal region in generalization, Daphna Shohamy and Anthony Wagner studied brain activity in healthy undergraduates with fMRI while using a variant on the Myers et al. acquired-equivalence procedure illustrated in [Figure 6.21](#). As shown in [Figure 6.23](#), they found that the magnitude of activation increase in the hippocampus (as well

as in the midbrain) during phase 1 training correlated with subsequent accuracy on the generalization trials in phase 3. Interestingly, they found no such correlation between hippocampal activation during the phase 3 generalization trials and the accuracy on those trials. Their finding that the critical hippocampal activity occurred during learning, not generalization, confirms Gluck and Myers's prediction that the hippocampal role in acquired equivalence is to transmit an appropriate (compressed or differentiated) stimulus representation during learning that permits subsequent use of this learning in a broader, more flexible range of future transfer situations.



correlated with accuracy in tests of generalization across all subjects in the Shohamy and Wagner study.

Data from [Shohamy & Wagner, 2009](#).

Description

The illustration of the human brain highlights the hippocampus to denote activation. The scatterplot has horizontal axis labeled, percent correct on transfer generalization, ranging from 30 to 100, in increments of 10. The vertical axis is labeled, brain activation during learning, ranging from negative 1.0 to 1.0, in increments of 0.5. 23 plots on the graph show positive correlation and the regression line increases from (38, negative 0.4), and ends at (100, 0.4). All 23 plots are scattered along the regression line.

Other Studies of Generalization in Schizophrenia

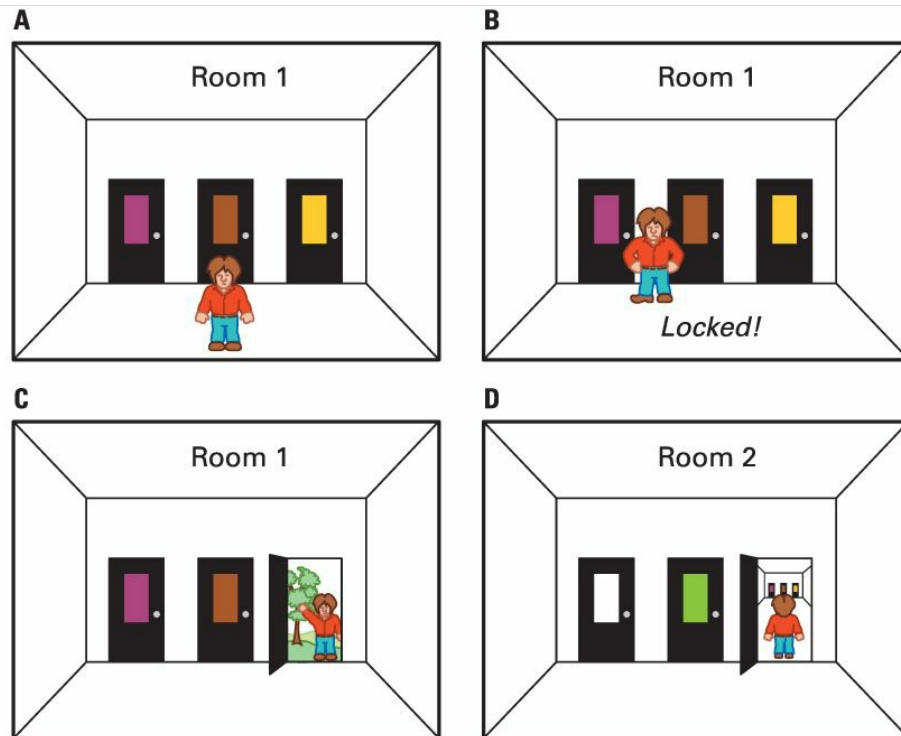
Acquired equivalence is not the only task that has been used to study the role the hippocampal region plays in generalization for people with schizophrenia. Other studies of people diagnosed with schizophrenia have shown that they are impaired at transitive inference, a form of hippocampal-region-dependent reasoning in which learned information is used to guide later inferences. If you learn that Liz is smarter than Alice and Alice is smarter than Isabel, then by transitive inference, you would predict that Liz is smarter than Isabel. Using “>” to represent “is smarter than,” we would write this inference as “if Liz > Alice and Alice > Isabel, then Liz > Isabel.”

In a study of this kind of learning and transitive inference, patients and controls were trained on a series of learned discriminations that were hierarchically organized according to the pattern A > B, B > C, C > D, and D > E ([Titone, Ditman, Holzman, Eichenbaum, & Levy, 2004](#)). They were then tested on each of these four training pairs, as well as on two novel “inference” pairs. The novel pairs, consisting of stimuli the participants had seen before but that had not previously been paired, were as follows: (1) A ? E, which can be evaluated (i.e., should “?” indicate “<” or “>”?) without consideration of hierarchical relations (because A and E each only appear in one type of relationship, superior or inferior to another cue), and (2) B ? D, which can only be evaluated by hierarchical relations because both B and D have previously been shown in superior and inferior relationships to other cues.

Patients and controls successfully learned the training pairs and correctly responded to the nonrelational A ? E pair. However, the patients were less accurate than controls in responding to the relational B ? D pair, consistent with the hypothesis that higher-level memory processes

associated with relational memory organization (and with its use in generalization from training to transfer tasks) are impaired in schizophrenia.

Kéri and colleagues have also studied generalization in schizophrenia using a task in which participants navigate a cartoon character, Kilroy, through a sequence of four rooms by learning to choose the unlocked door from among three colored doors in each room. In the training phase, each stimulus leading to reward (the unlocked door in each room) is trained via feedback until the complete sequence is learned ([Figure 6.24](#)). Note that in each new room in the experiment, the colors change on the doors, and it is door color, not door location (left, right, or middle), that is the cue to be learned. In the generalization phase, the decision-making context is manipulated so that in a given room, a door whose color was correct in another room has a different color in the new room and therefore is not the correct door in the new room. Compared with controls, patients diagnosed with schizophrenia were markedly impaired at transferring previously learned rules to new rooms where the options were different, even when they had successfully mastered the initial training phase ([Polgár et al., 2008](#)). This narrow rigidity in applying past experiences to understanding the future may help shed some light on why people with schizophrenia often have difficulty learning which relationships in the world are or are not reliable and truly predictive of reality.



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FIGURE 6.24 The chaining task used to study learning and generalization in schizophrenia Top left: At the start of the experiment, participants choose among the closed doors in room 1. Top right: Choosing the purple door is incorrect and results in no exit. Bottom left: Choosing the yellow door in room 1 is correct, and Kilroy goes outside. Bottom right: In room 2, the participant chose the correct yellow door (on right), allowing Kilroy to exit back into room 1.

Description

Clockwise from top left, the cartoons are as follows. A, the man is in room 1 with doors purple, brown and yellow from left to right. B, the man is in room 1 with doors purple, brown and yellow from left to right. The man is near the purple door and a label reads, locked. C, the man is outside room 1, through the open yellow door. and D, the man has entered room 1 by opening the third door from left in room 2. The other doors in room 2 are white and green, from left to right. He faces the three doors in room 1.

Altered Generalization in Autism Spectrum Disorder

A stimulus such as a large blue circle has at least three features by which it can be recognized: size, color, and shape. In short, a large blue circle is actually a compound stimulus, similar to the tone-and-taste, tone-and-light, or tone-and-shock combinations described in the [Chapter 4](#) discussion of compound conditioning. [Chapter 4](#) shows how learning about such stimuli can be

understood as learning associations to the various component features of the stimulus compounds. In learning trials using a large blue circle, assuming all three features are equally salient, we would expect some learning to accrue to all three features. Later, if a new object is presented that shares just one of these features but not the others, we would expect the learning to generalize at least a little bit to the new object. This is indeed what happens when most people learn about compound stimuli. But it is not what we see when children with autism spectrum disorder (ASD) learn and generalize.

ASD is a lifelong neurodevelopmental condition for which there is no known cure. It differs greatly from person to person in severity, is most commonly associated with poor social skills and impaired communication abilities, and has no single neural locus. Rather, anatomical and functional abnormalities have been found in the prefrontal cortex, basal ganglia, temporal lobes, and limbic regions. Many studies have suggested that the variability in ASD may be associated with variations in altered (especially reduced) connectivity within and between these brain regions.

In a landmark study conducted over 40 years ago, researchers identified “stimulus overselectivity” as a key cognitive impairment in ASD. In this study, autistic children were trained with a successive discrimination task, essentially being asked to respond with “Go” or “No go” to a series of compound stimuli. Each of the compound stimuli had four simultaneous components: a 150-watt red light (visual), a 63-decibel white noise (auditory), an inflatable blood-pressure cuff (tactile), and a variable period during which these cues were presented (temporal). After the children learned to respond appropriately with “Go” whenever they were presented with this compound stimulus (and “No go” to all others), the various stimulus components were presented separately, and the response (or lack of response) to each was recorded.

In contrast to the control group of children without ASD, who usually learned to generalize on the basis of about three of the component features, children diagnosed with ASD learned in a hyperselective manner, focusing on one and only one of the different features. There was, however, no consistent preference among the children with ASD for any particular modality. One child might focus on the visual stimulus, another on the auditory stimulus, and a third on the tactile stimulus. It was as if the children with ASD had a hyperfocused attention that picked just

one feature and tuned out all others.

Some people have referred to this inability to process compound cues as a form of “tunnel vision” in which the children with ASD cannot integrate multiple sources of information and instead become highly selective, adhering to just one ([Ploog, 2010](#)). It remains unclear, however, whether this attentional deficit in ASD is due to some kind of sensory overload preventing the children from processing more than one feature or whether their attention somehow becomes fixed on the first feature they notice and they fail to disengage from that to notice the others. The degree to which children with ASD exhibit stimulus overselectivity in generalization tasks has been linked to a variety of clinical behaviors associated with ASD, including impaired social skills, language deficits, lack of inhibitory control, and ritualistic behaviors ([Ploog, 2010](#)). This finding opens up the tantalizing possibility, still to be tested, that if cognitive deficits in stimulus overselectivity could be remediated through training or other treatments, children with ASD might experience broad improvements in a range of clinically significant impairments that hamper their quality of life and ability to socialize.

Interim Summary

- People with schizophrenia show deficits in generalization, consistent with their impaired hippocampal-region function.
- Generalization deficits in acquired equivalence in people with schizophrenia are probably linked to impaired hippocampal function during learning, as shown by functional brain-imaging studies.
- Children with autism spectrum disorder have abnormal patterns of generalization based on hyperfocusing on just one feature and tuning out all others.

Synthesis

Since we last saw her, 2-year-old Sabrina has wiggled out of her high chair, walked across the kitchen, and sneaked out the back door. While exploring the backyard, she comes across a small brown dog blocking her path. It turns to Sabrina and begins to growl. Sabrina has never seen this dog before. He looks something like her own friendly dog, Max, of whom she is quite fond, but this dog is new to her. Should Sabrina approach the dog or flee?

Sabrina's visual cortex registers the visual information about the animal's brown fur, and her auditory cortex picks up the growling sound. Because her dog, Max, is brown, the brown-detecting regions of Sabrina's visual cortex are highly sensitive and enlarged and can quickly detect and recognize Max when he comes into the room. On the basis of this dog's similar color to Max, she is disposed to generalize her positive feelings toward Max to this new dog and view him as friendly. At the same time, Sabrina's auditory cortex is highly attuned to Max's unique bark, and this dog sounds less like Max and more like the snarling and scary dogs she sometimes sees in the park. On the basis of bark, she is inclined to view this dog as being different from Max and potentially dangerous. In sum, the information from her visual cortex is in conflict with that from her auditory cortex: one suggests generalization and the other discrimination.

However, at the same time as her primary sensory cortices are processing the sights and sounds associated with this new dog, Sabrina's hippocampal region is combining the visual and auditory information with other information about the overall context in which this event is occurring: the backyard of her home on a warm summer day. The combination of this contextual information and the visual and auditory cues picked up from the animal allows Sabrina's hippocampus to recognize an important pattern: her father's friends often visit during the summer with their dogs. Based on this configuration of local contextual information (the place and the season) and the sight and sound of the animal, Sabrina infers that this particular dog is more likely to be friendly than aggressive. She reaches out and pets him, and he rolls over to let her scratch his belly. Your ability to discriminate and generalize, to recognize friend from foe—like Sabrina's—will govern your choices and determine what you learn from each experience.

Chapter 6 Review

Know Your Key Terms

[acetylcholine \(ACh\)](#)
[acquired equivalence](#)
[category](#)
[concept](#)
[concept formation](#)
[confirmation bias](#)
[consequential region](#)
[discrete-component representation](#)
[discrimination learning](#)
[distributed representation](#)
[errorless discrimination learning](#)
[generalization](#)
[generalization gradient](#)
[hippocampal region](#)
[inductive inference](#)
[medial temporal lobe](#)
[negative patterning](#)
[nucleus basalis](#)
[prototype](#)
[sensory preconditioning](#)
[stereotype](#)
[stimulus control](#)
[stimulus representation](#)

Quiz Yourself

1. After training in which a single stimulus (such as a light of a particular color) has been reinforced repeatedly, _____ around that trained stimulus show a peak, or point of maximal responding, corresponding to the original stimulus on which the animal was trained. ([p. 229](#))

2. Discrete-representation learning models are useful for describing how organisms learn about highly _____ stimuli, such as a tone and a light, but they do not work as well with stimuli that have some inherent _____, such as lights of different colors. (p. 233)
3. In Thorndike's view, yellow and yellow-orange are not two totally separate stimuli. Rather, he argued, they are each composed of many distinct elements, some shared and others not shared. Ideas much like this were promoted by W. K. Estes in his _____ theory and by David Rumelhart and colleagues in their _____ models. (p. 235)
4. Two objects or substances that initially seem very similar, or even indistinguishable, eventually come to be distinguishable when each is repeatedly paired with a different label, name, or outcome. This is an example of _____. (p. 239)
5. A gradual transition from easy to hard discrimination tasks so as to all but eliminate errors during learning is called _____. (p. 242)
6. The _____ procedure shows that co-occurrence of two stimuli is sufficient to produce generalization from one stimulus to another. (p. 244)
7. Negative patterning is just one example of a larger class of learning phenomena that involve configurations of stimuli and that cannot be explained using _____. (p. 248)
8. _____ are internal psychological, or mental, entities constructed on the basis of our experiences with the world. In contrast, _____ are a feature of the world, a class or division of people or things that have some shared characteristics. (p. 249)
9. Many primary sensory cortical areas are organized _____, in that each region responds preferentially to a particular type of stimulus, and neighboring regions respond to similar stimuli _____. (pp. 257–258)
10. One key brain region that determines whether a stimulus merits cortical remapping is the _____. (p. 262)

11. Animals with hippocampal damage are impaired at tasks that require sensitivity to stimulus–stimulus relationships such as _____ and _____. (pp. 263–264)
12. People with schizophrenia show abnormalities in the shape of the _____, most notably an overall smaller volume. (p. 267)
13. Although some of our stereotypes are based on personal encounters with members of another group, many stereotypes are acquired through _____. (p. 252)
14. Rosch argued that natural categories are adopted when they provide us with value in improving our ability to make _____ inferences. (p. 253)
15. Confirming Gluck and Myers’s prediction of the hippocampal role in acquired equivalence, Daphna Shohamy and Anthony Wagner found that critical hippocampal activity occurs during _____ rather than during _____. (p. 269)
16. Lesions of the entorhinal cortex (within the hippocampal region) eliminated _____ in rabbit eyeblink conditioning. (pp. 264–265)
17. Upon activation, the nucleus basalis neurons release _____, a neurotransmitter that promotes neuronal plasticity. (p. 262)
18. Richard Thompson’s experiments in the 1960s suggested that animals _____ and have difficulty discriminating various stimuli when their primary sensory cortex is damaged. (p. 261)
19. If you are asked to think about a typical cat, you are likely to think of a whiskered creature with four legs that would enjoy eating meat. This abstract representation of the idealized or typical member of a category is called the category’s _____. (p. 251)

Concept Check

Consider the following experiment, conducted by Robert Rescorla in 1976. In phase 1, he trained rats to associate a yellow light (the CS) with a US ([Rescorla, 1976](#)). After all the rats

were fully trained and giving reliable CRs to the yellow light, he divided the animals into two groups. Rats in the experimental group received a second phase of training with an orange light as the CS until they learned to give reliable CRs. The control rats, however, continued to be trained with the yellow-light CS. Finally, in the third test phase of the experiment, all animals were exposed to a yellow light as the possible CS.

1. What do you predict occurred in phase 3: did the rats in the experimental group give a larger or smaller response to the yellow light compared with the rats in the control group?
2. Why? And what does this suggest about the best way for you to prepare for your tennis tournament?

Hint: In keeping with the shared-elements approach of Thorndike and Estes, designate X as a shared element common to both yellow lights and orange lights (capturing the similarity of these two cues) and designate Y and O as the elements unique to yellow and orange lights, respectively. Thus, the yellow light can be viewed as a compound cue YX, and the orange light can be viewed as a compound cue OX. Using this shared-elements representation for these two similar stimuli, apply the Rescorla–Wagner model to each of the three phases of the experiment and predict which group should give a stronger response to the yellow light in phase 3.

Answers to Test Your Knowledge

Discrete-Component or Distributed-Representation Models?

1. A discrete-component model is sufficient because all the tones are predictive of shock, and there is no need to discriminate among them.
2. A distributed representation would be required because the model needs to have a way to capture the degree to which green is more or less similar to blue and red.

Discriminating Between Generalization Paradigms

1. d (negative patterning). Candy → good, flowers → good, candy + flowers → bad.

2. b (sensory preconditioning). Phase 1: deep voice + beard. Phase 2: beards → strong. Phase 3: infer deep voice → strong.
3. a (discrimination training). Music1 → Brahms, music2 → Schubert.
4. c (acquired equivalence). Mark → food1, Kaori → food1; Mark → music1, infer Kaori → music1.

Cholinergic Neurons and Learning

Since the nucleus basalis promotes discrimination learning by altering cortical mapping, discrimination memories that were acquired before the lesion will be unaffected. However, any attempts to teach discrimination learning after surgery will not be effective, as cortical plasticity is no longer possible.

Memory Module

Chapter 7 Episodic Memory and Semantic Memory Memory for Events and for Facts



Mr Elliott Neep/Getty Images

Behavioral Processes

[Features of Episodic Memory and Semantic Memory](#)

[Encoding New Memories](#)

[Retrieving Existing Memories](#)

[*Learning and Memory in Everyday Life: Extraordinary Memorizers*](#)

[When Memory Fails](#)

[Memory Consolidation and Reconsolidation](#)

[Metamemory](#)

[*Learning and Memory in Everyday Life: Optimize Your Study Habits*](#)

Brain Substrates

[Cortical Networks for Semantic Memory](#)

[The Medial Temporal Lobes and Memory Storage](#)

[Determining What Gets Stored](#)

[Long-Term Storage and Retrieval](#)

Clinical Perspectives

[Transient Global Amnesia](#)

[*Learning and Memory in Everyday Life: The Cost of Concussion*](#)

[Functional Amnesia](#)

FOR OVER 50 YEARS, THE IDENTITY OF THE MAN KNOWN ONLY BY THE INITIALS H.M. was one of the most closely guarded secrets in psychology.

H.M.'s troubles began in childhood, with epileptic seizures during which the neurons in his brain fired wildly and uncontrollably. By age 16, the seizures were frequent and debilitating. Severe attacks, during which he might convulse and lose consciousness, occurred weekly; minor attacks occurred up to 10 times a day. H.M. struggled to complete high school and finally graduated at age 21, but the seizures were so frequent and so severe that he had difficulty holding a simple job. His doctors put him on a near-toxic diet of anticonvulsant drugs, but still the seizures continued.

In 1953, in desperation, H.M. and his family agreed to try brain surgery. At the time, doctors knew that many epileptic patients experience seizures that start in either the left or right hemisphere, usually in the [medial temporal lobes](#), the inner (or medial) surfaces of the temporal lobes. Doctors had found that surgical removal of the medial temporal lobe from the hemisphere

where the seizures originated could often eliminate the source of the problem and cure the epilepsy in these patients. Because H.M.'s seizures were so severe, and because their precise origin could not be determined, the doctors decided to remove his medial temporal lobes bilaterally ([Corkin, Amaral, Gonzalez, Johnson, & Hyman, 1997](#)).

medial temporal lobes

The medial (or inner) surface of the temporal lobes that contains the hippocampus, the amygdala, and other structures important for memory.

Medically, the operation was a success: H.M.'s seizures declined drastically in frequency and severity. But there was a terrible cost. H.M. was left with [amnesia](#), or severe memory impairment. Specifically, he lost the ability to form new memories for facts and events ([Scoville & Milner, 1957](#)). He could no longer remember what he had eaten for breakfast or why he was in the hospital. He could spend all morning working intensively with a psychologist and then take a break for lunch; an hour later, he would not recognize the psychologist at all ([Haglund & Collett, 1996](#)). When H.M. found out that a favorite uncle had died, he experienced intense grief—and then forgot. Again and again, he asked after the uncle and reacted with surprise and fresh grief every time he was told of the death ([Milner, 1966](#)). H.M. himself was painfully aware of his poor memory and described his life as constantly waking from a dream he could not remember ([Milner, Corkin, & Teuber, 1968](#)).



"Henry Molaison, aged 60, at MIT in 1986.
Photograph by Jenni Ogden, first published
in her book "Trouble in Mind: Stories from a
Neuropsychologist's Casebook."

Henry Molaison, known to brain researchers everywhere as H.M., is seen here in 1986 at age 60, in the neuropsychology test laboratory at Massachusetts Institute of Technology, Cambridge, Massachusetts. How does the finding of severe memory loss in a patient with localized brain damage challenge Lashley's view of how memory is stored in the brain?

amnesia

Severe memory impairment.

Despite his devastated memory, H.M.'s personality was basically unchanged, and after the

operation, his IQ actually went up—probably because without constant seizures, he could now concentrate better on whatever he was doing. He could not follow the plot of a television show because the commercials would interrupt his memory of the story line, but he could still amuse himself solving crossword puzzles. As long as H.M. paid attention to a task, he could perform well; as soon as he turned his attention to something else, the information vanished. H.M. was living proof that the ability to form new fact and event memories depends on the medial temporal lobes but that many other kinds of memory (and cognitive function) do not.

On December 2, 2008, H.M. passed away, and, by prior agreement, the details of his identity were released to the public. Memory researchers finally learned the name—Henry Gustav Molaison (pronounced “Mollisson”)—and saw the face of the man who taught us so much. Even in death, H.M. continues to contribute. His brain was donated to science, and a team of researchers digitized images of the preserved tissue to create a “virtual” map for others to study.

The studies in which H.M. participated revolutionized scientific thinking about memory. Before H.M., many scientists held to Karl Lashley’s view, described in [Chapter 2](#), that memory is encoded in a distributed fashion across the brain. H.M.’s case showed that the formation of new fact and event memories depends on distinct brain regions, separate from the brain regions that mediate other cognitive functions.

Fortunately, amnesia as severe as H.M.’s is extraordinarily rare. But amnesia does occur, sometimes as a result of a brain injury and sometimes as a result of disease or extreme stress. Some patients, like H.M., lose the ability to form new memories. In other cases, patients lose memories of a specific past event or—rarer still—lose all memories of their identity and personal history. Cases such as these remind us that our memories—the facts we know and the events we remember—define us.

7.1 Behavioral Processes

Think back to the day of your high school graduation. Where was the ceremony held? Who sat near you? What were you wearing? Did the school band perform? What were your feelings—pride, excitement, or perhaps impatience for the ceremony to end so you could celebrate with your friends?

These details of your graduation constitute an [episodic memory](#), which is a memory for a specific event in your life ([Tulving, 1972, 1983, 2002](#)). An episodic memory includes information about the spatial and temporal context in which it was formed—that is, where and when the event occurred.

episodic memory

Memory for personal experience of specific autobiographical events; it includes information about the spatial and temporal contexts in which the event occurred.

Related to, though distinct from, episodic memories are [semantic memories](#), which are memories for facts and general knowledge about the world, as well as personal information such as your own name and your favorite food. Unlike episodic memory, semantic memory need not include information about the time and place where the information was acquired. For example, if asked to name the first president of the United States, or to state your mother's maiden name, you probably know the answers. But you may not remember when and where you learned either piece of information. Whereas episodic memory is what we *remember*, semantic memory is what we *know* ([Tulving, 1985](#)).

semantic memory

Memory for facts or general knowledge about the world, including general personal information.

Features of Episodic Memory and Semantic Memory

Episodic and semantic memory share two key features (see [Table 7.1](#)): (1) they can be communicated flexibly, and (2) they are accessible to our consciousness. Flexible communication means we can communicate our episodic and semantic memories in formats different from the way they were originally acquired. When you remember a particular episodic memory—say, the memory of your graduation—you can describe the details you recall, even if

you have never tried putting them into words before. Similarly, if someone were to show you a photo of your graduation, you would probably be able to recognize the scene, even if you had never seen it from that vantage point.

TABLE 7.1 Comparing and contrasting episodic and semantic memory

Episodic memory	Semantic memory	Same (✓) or Different (X)
<i>Event related: “I remember”</i>	<i>Factual: “I know”</i>	X
Can be <i>communicated flexibly</i> —in a format other than that in which it was acquired	Can be <i>communicated flexibly</i> —in a format other than that in which it was acquired	✓
<i>Consciously accessible</i> (you know that you know)	<i>Consciously accessible</i> (you know that you know)	✓
Attached to a <i>spatial and temporal context</i>	<i>Not necessarily</i> attached to a spatial or temporal context	X
You must have experienced the event <i>personally</i>	Can be <i>personal or general</i> information	X
Learned in a <i>single exposure</i> ; can be weakened by exposure to similar events	Can be learned in a single exposure but can also be <i>strengthened by repetition</i>	X

As for semantic memory, if someone asks you how to get from the library to the cafeteria, you can answer by giving verbal directions or by drawing a map, even if you have never attempted to put the information into these specific formats before. Similarly, after memorizing a list of historical facts, you can communicate that knowledge on an exam whether the format is true/false, multiple-choice, or essay questions.

In contrast to episodic and semantic memory, some other kinds of memory are hard to communicate in ways other than how they were originally learned. For example, [Chapter 8](#) discusses perceptual-motor skills, such as tying your shoes. You can probably tie a shoe easily, but imagine if someone asked you for a short description of how to do it. Odds are you would find it difficult to comply—and you might even have to go through the hand movements to remind yourself what they are. Skill memories are generally not easy to communicate in new ways.

Because of these similarities between episodic memory and semantic memory, some researchers use the broader term **declarative memory** to include both episodic and semantic memory, reflecting the fact that it is easy to verbalize (“declare”) or otherwise communicate your knowledge ([Anderson, 1976](#); [Cohen & Squire, 1980](#); [Squire, Knowlton, & Musen, 1993](#)). Other

kinds of memory—grouped under the heading **nondeclarative memory**—are not always easy to communicate verbally ([Squire & Knowlton, 1995](#)). Skill learning is one kind of nondeclarative memory, as are classical and operant conditioning.

declarative memory

A broad class of memories, both semantic and episodic, that can typically be verbalized (“declared”) or explicitly communicated in some other way.

nondeclarative memory

A broad class of memory that includes skill memory and other types of learning that do not fall under either the episodic or semantic memory categories and that are not always consciously accessible or easy to verbalize.

Other researchers prefer the term **explicit memory** ([Graf & Schacter, 1985](#); [Schacter, 1987](#)) in discussing episodic memory and semantic memory to reflect the fact that both are consciously accessible, or “explicit” (you know that you know). By comparison, **implicit memory** is memory that you may not be aware you have acquired. For example, H.M. (who could not acquire new episodic or semantic memories) could learn new skills such as reading mirror-reversed text ([Gabrieli, Corkin, Mickel, & Crowden, 1993](#)). Just like healthy controls, H.M.’s performance slowly improved with practice. But when asked about these new skills, H.M. reported having no conscious memories of the training sessions and no knowledge of his new skill. Thus, this learning was implicit.

explicit memory

A category of memory that includes semantic memory and episodic memory and that consists of memories of which the person is aware: you know that you know the information.

implicit memory

Memory that occurs without the learner’s awareness.

H.M. is a special case, of course. Most of us, while practicing new skills, will also form consciously available episodic and semantic memories of what we are learning. In fact, it has proven extraordinarily difficult to disentangle explicit and implicit learning in healthy adults ([Shanks, 2010](#)). H.M.’s case demonstrates that conscious awareness is not absolutely *necessary* for implicit memories to form.

What Distinguishes Episodic Memory from Semantic Memory?

Despite their similarities, episodic memory and semantic memory have three contrasting properties ([Table 7.1](#)). First, as stated previously, episodic memories concern *specific events that*

occurred at a particular place and time: you must remember when and where you experienced those events. Semantic memories involve *factual information*: you need not remember where and when you learned this information—only the fact itself. Second, episodic memory is *always autobiographical*, based on your *personal experiences*. Semantic memory *can be autobiographical*, but it need not be. Third, episodic memory is always acquired in a *single exposure*—the event itself. Semantic memory can be acquired in a single learning event, but it need not be.

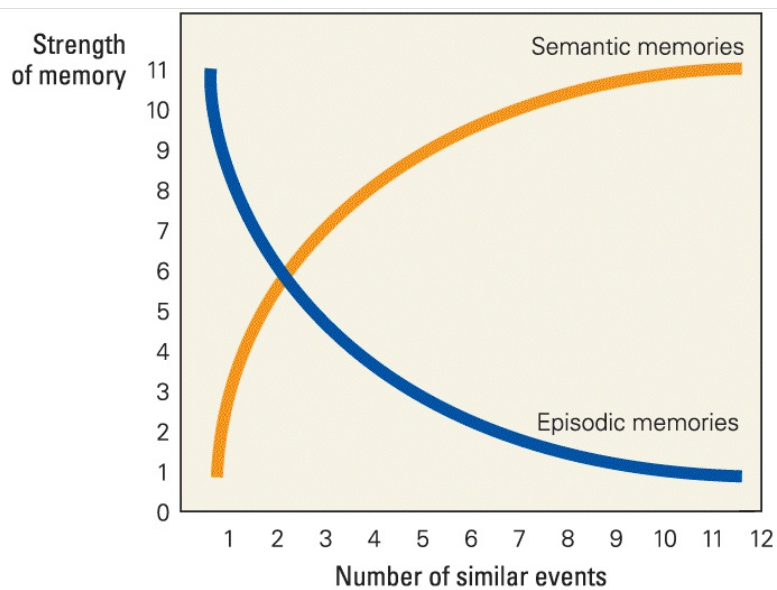
For example, you can have an episodic memory of a specific event in your life, such as your graduation or your first kiss. You can also have an episodic memory for how you learned of an important event, such as the death of a family member, even if you were not actually present; for example, you may remember exactly where and when you received a phone call with the news. But the episodic memory is always about remembering your personal experience of the event, including how you felt at the time. In contrast, semantic memory can include autobiographical information (such as your mother's name, where you were born, or your favorite flavor of ice cream), but it is information available to you as a known "fact," analogous to the names of the planets in the solar system, the meaning of the word *dog*, or the atomic weight of boron.

Some memories straddle the line. Suppose you participate in a laboratory experiment that takes place over 3 days. On day 1, you are asked to memorize a list of familiar words (DOG, CHAIR, CLOUD, etc.). On day 2, you are given a second list to memorize (CAT, TABLE, STAR, etc.). Finally, on day 3, the experimenter asks you whether a specific word (CHAIR) was on the first list or the second list. Here, you have to remember the specific event in which you saw that word—in the testing lab, on the first day of the experiment—as distinct from all the other times in your life when you have seen that word. Thus, your memory of learning the word CHAIR on study day 1 is an episodic memory, "tagged" with a time and place. On the other hand, suppose the experimenter sends you home to study the two lists. Every night before bed, you take out the lists and work on memorizing the words. A week later, you return to the lab for your memory test. Now, you may have acquired a semantic memory of which words were on which list, but you may not distinctly remember all the individual study sessions that helped you acquire the information. In this case, your memory of the list items would be semantic memory.

This example also illustrates the third difference between episodic memory and semantic

memory: the fact that episodic memory is acquired in a single exposure, the event itself. In principle, semantic memories can be acquired in a single exposure, too, particularly if the information is sufficiently interesting or important. For instance, it might take you several exposures to memorize the Latin word for “arch”—*fornix*—unless you are also told that, in ancient Rome, prostitutes used to ply their trade under arches, which is where we get the modern word “fornicate.” Such extra information, which relates the vocabulary item to other information you know, may help you remember the word after only a single exposure.

But, often, semantic information needs multiple exposures before being fully acquired. So, for example, you may have to study a Latin vocabulary list several times before you have all the items memorized. In general, repeated exposures to a single fact *strengthen* semantic memory for that fact ([Linton, 1982](#)); by contrast, repeated exposure to very similar events may *weaken* episodic memory for any one of those events ([Figure 7.1](#)). If you park your car in the same large parking lot every day, you may confuse the episodic memories of all the prior, highly similar parking events, making it hard to remember exactly where you parked the car today. This is one reason why any large parking lot contains a number of people walking around with panicked expressions on their faces.



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

FIGURE 7.1 Episodic memory and semantic memory In general, semantic memory is strengthened by repetition, but episodic memory can be weakened by repeated exposure to similar events.

Description

The horizontal axis shows the Strength of memory. The vertical axis shows the number of similar events. The data in the graph are as follows. The line for semantic memories indicates increasing strength of memory with repetition of similar events. The line for episodic memories indicates decreasing strength of memory with repetition of similar events.

Which Comes First: Episodic Memory or Semantic Memory?

The exact relationship between episodic memory and semantic memory is a matter of intense debate. The distinction between the two was first made by psychologist Endel Tulving, who argued that episodic memory grows out of semantic memory ([Tulving, 2002](#)). According to this view, an organism has to have a certain amount of semantic information before episodic memories can be built on that framework. If you do not know what a graduation is, you can hardly have an episodic memory for any specific graduation—even your own.

But current research is pointing to the idea that semantic memory represents information we have encountered repeatedly—often enough that the actual learning episodes are blurred, and only the semantic memory (or “gist”) remains ([Conway, 2009](#); [Winocur & Moscovitch, 2011](#)). For example, if you remember the very first time you learned about George Washington, then you have an episodic memory for that event—perhaps it was a history class. But if you have heard about George Washington in many different classes and have also read about him in books and seen television portrayals, then you have accumulated a general store of knowledge about the first U.S. president, whether or not you remember the individual episodes.

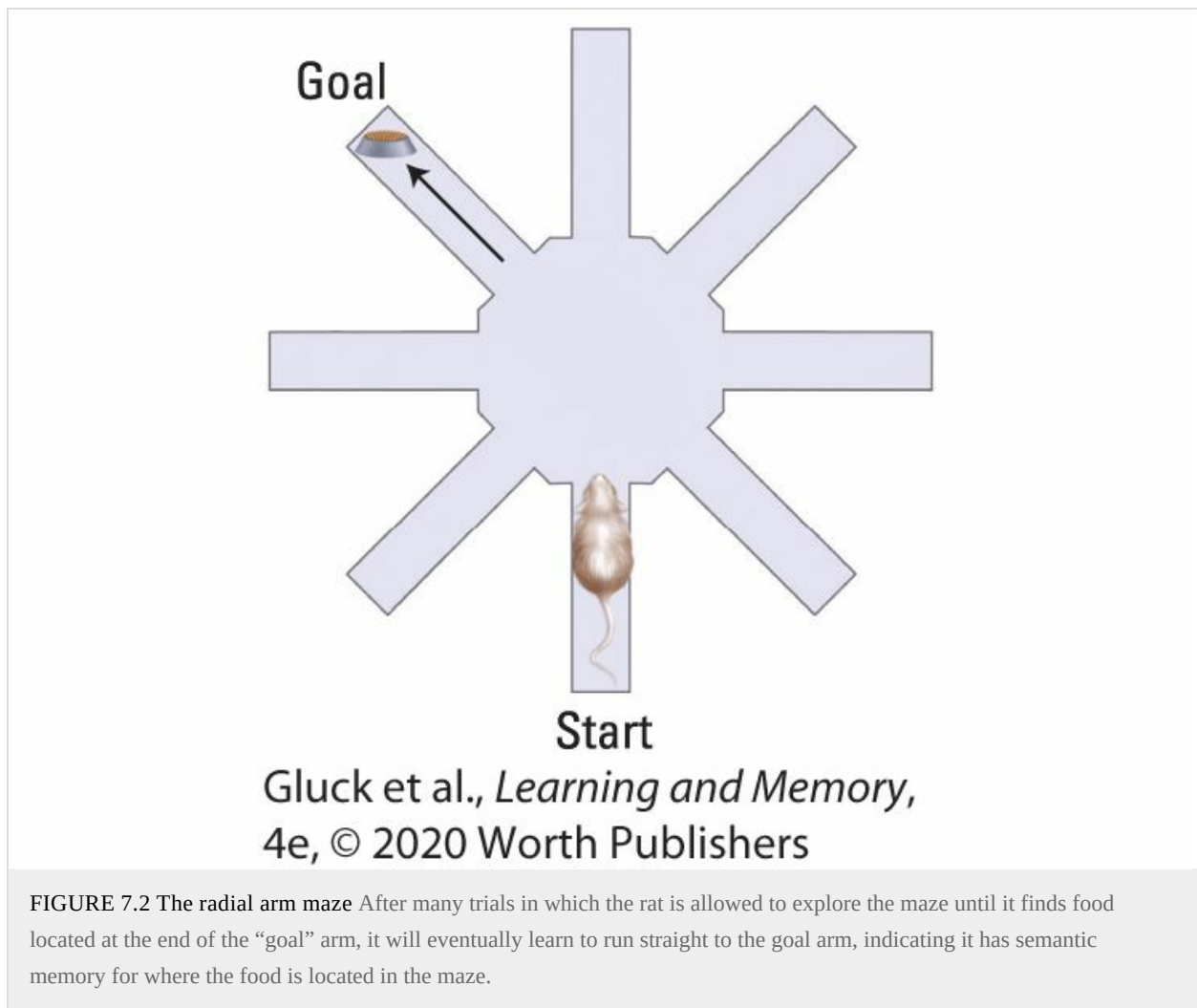
Similarly, whereas you can probably remember your graduation, you may not have an episodic memory of your first birthday, in the sense of being able to “mentally time travel” and relive the episode. But if you have heard the story many times and seen family photos of your infant face happily smeared with chocolate icing, then you may have a strong and highly detailed semantic memory for the party. In this case, the semantic memory is information you *know* about your past history rather than an episodic memory that you can *remember*.

Can Nonhumans Have Semantic Memory?

The easiest way to assess semantic memory in humans is through question-and-answer. If an

experimenter asks you the name of the first U.S. president and you reply “George Washington,” then the experimenter can safely conclude that you have a semantic memory of that fact. Things get a little more problematic with nonhuman animals; we cannot ask a rat to name the president. But animals do learn semantic information and can communicate it flexibly—in ways different from how the information was originally acquired ([Eichenbaum, 2004](#)).

For instance, [Figure 7.2](#) shows a *radial arm maze*: viewed from above, the maze has a central area from which several arms branch off like the spokes of a wheel. The top of the maze is open so that a rat placed in the maze can see out and use landmarks in the room, such as windows or posters, to help navigate.



Description

The illustration shows a circular model of a radial arm maze. It consists of a larger central room and eight arms. A model of a rat kept on one of the arms begins to run through the arms of the maze. One arm is labeled Start, and the other is labeled Goal.

A radial arm maze can be used to assess many kinds of learning and memory. In one version, researchers put food at the end of one arm (the “goal” arm) and place the rat at the end of a different arm (the “start” arm). The rat is then allowed to roam the maze until it finds and consumes the food. Over many such trials, the rat learns where the food is located, so when it is placed in the start arm, it will eventually run directly to the goal arm. If a well-trained rat is placed in a new start arm and still runs to the food—via a route it has never used before—then researchers conclude that the rat “knows” where the food is and can use this information flexibly in new ways (such as using a new route to get there). Thus, most researchers would agree that this is an example of semantic memory in rats.

Other examples of semantic memory in animals are described in [Chapter 6](#)—such as the ability of trained pigeons to correctly classify music or paintings as belonging to particular genres. To the extent that the pigeons have learned the features associated with each genre and are able to apply that learning flexibly when presented with novel exemplars, that learning would also qualify as semantic memory.

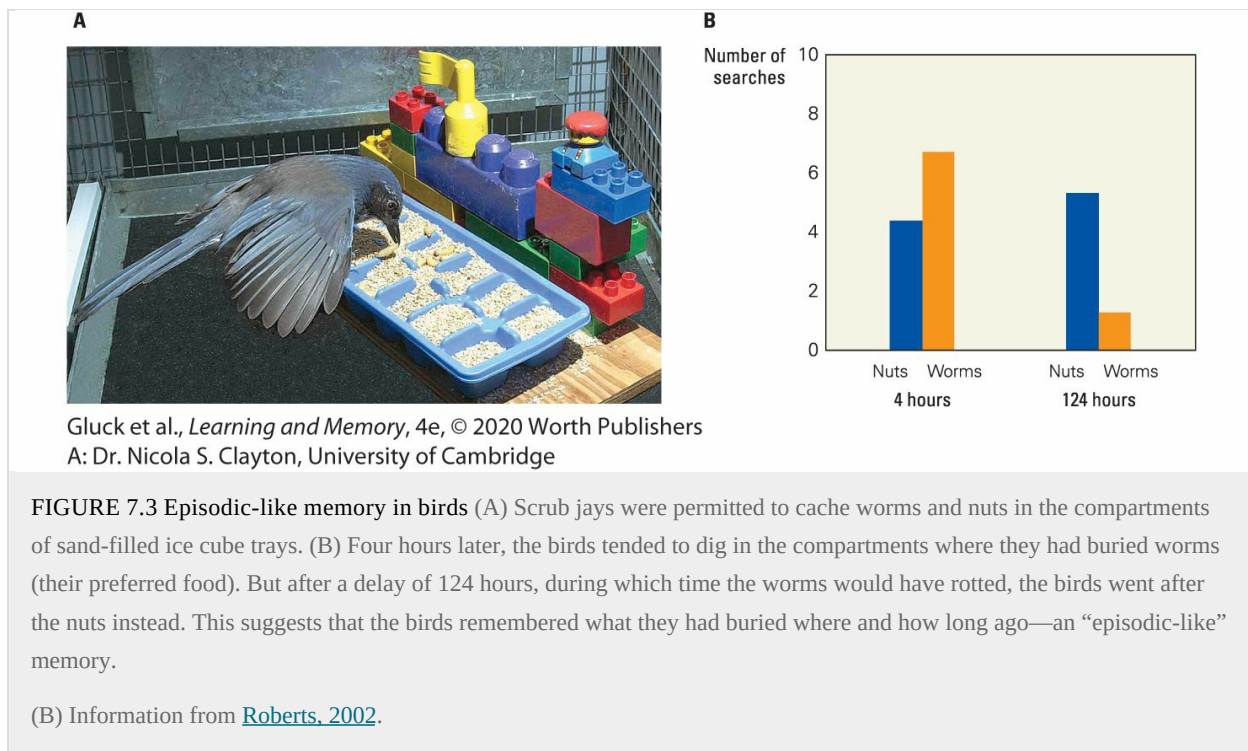
Can Nonhumans Have Episodic Memory?

Episodic memory in animals is harder to assess than semantic memory. In fact, some researchers have explicitly argued that animals cannot maintain episodic memories, at least not in the way that humans do ([Roberts & Feeney, 2009](#); [Tulving, 2002](#)). Tulving, in particular, argued that episodic memory requires mental time travel, a re-experiencing of the event in memory; this in turn requires a conscious sense of self, as well as a subjective sense of time passing, which—Tulving claimed—have not been demonstrated in nonhumans ([Tulving, 2002](#)). But other researchers argue that there is mounting evidence of animals forming “episodic-like” memories for specific events, including information about the spatial and temporal contexts in which those events occurred ([Clayton, Yu, & Dickinson, 2001](#); [Crystal, 2010](#)).

For instance, gorillas seem to remember specific autobiographical events, and they can flexibly communicate information about these events to human testers. One gorilla, King, was taught to

“name” various fruits and humans by using cards with drawings that represented the fruits and humans ([Schwartz, Colon, Sanchez, Rodriguez, & Evans, 2002](#)). This general knowledge about how to use the cards qualifies as semantic memory. Researchers then attempted to assess whether King could remember distinct events he had experienced. During the day, King received various pieces of fruit from different human handlers. Twenty-four hours later, when asked (via the cards) who had given him a particular fruit the day before, King could use the cards to name the correct human. Because King had eaten several fruits and interacted with other humans during the course of several days, his performance seems to demonstrate that he had episodic-like memory for the events of the prior day—remembering not just that he ate fruit but the specific type of fruit, who gave it to him, and approximately when this happened. And he could communicate this behavior to the experimenters, using abstract symbols on cards. This seems to satisfy the criteria for episodic memory ([Schwartz & Evans, 2001](#)).

Birds may also be able to remember specific events and how long ago they happened. Scrub jays, for example, bury extra food in caches so they can retrieve it later. These birds accurately remember their cache locations and will return to those locations later, even if an experimenter has secretly removed the food in the meantime (to ensure that the birds cannot use scent to locate the buried food). To test episodic-like memory, Nicola Clayton and her colleagues allowed scrub jays to cache worms and nuts in sand-filled compartments of an ice cube tray ([Figure 7.3A](#)). The birds were permitted to look for the food either 4 hours or 124 hours (about 5 days) later. Normally, scrub jays prefer worms to nuts, and when tested after a 4-hour interval, the birds tended to dig in the compartments where they had buried worms ([Figure 7.3B](#)). But after a 124-hour interval, during which the worms would have rotted, the birds instead tended to dig for nuts, which would still be edible ([Clayton & Dickinson, 1999](#)).



Description

The scrub jay is seated on the compartments of the tray, filled with worms and nuts. A bar graph on the right has a horizontal axis, marking the Number of searches and vertical axis marking Nuts and for worms at 4 hours and again at 124 hours. The data of the graph are as follows. At 4 hours, the scrub jay search for nuts about 4 times and about 7 times for worms. At 124 hours, they searched nearly 6 times for nuts and only once for worms.

These results suggest that scrub jays can remember not only where they have stored food but what type of food was stored and how long ago ([Clayton et al., 2001](#); [Griffiths, Dickinson, & Clayton, 1999](#)). Rats, dolphins, and pigeons may have similar abilities to remember specific events, including when and where those events took place ([Kart-Teke, De Souza Silva, Huston, & Dere, 2006](#); [Mercado, Murray, Uyeyama, Pack, & Herman, 1998](#); [Zentall, Singer, & Stagner, 2008](#); [Zhou & Crystal, 2011](#)).

Some researchers go even further, arguing that some nonhuman animals (rats, dolphins, nonhuman primates, and some birds) do have a subjective sense of self and of time and that their behavior demonstrates that they can indeed mentally re-create events from their own past and imagine their own future ([Dere, Kart-Teke, Huston, & De Souza Silva, 2006](#); [Zentall, 2006](#)).

For the rest of this chapter, we will adopt the convention of referring to “episodic-like” memories

in nonhuman animals if those memories include what-where-when information about the spatial and temporal context in which the episode occurred ([Crystal, 2010](#); [Eacott & Easton, 2010](#); [Wang & Morris, 2010](#)). Use of the term *episodic-like* (rather than just *episodic*) acknowledges that we cannot directly ask nonverbal animals about their subjective sense of self or their ability to perform mental time travel. In the absence of direct evidence on these points, controversy continues over whether nonhuman animals can form true episodic memories or whether this ability is uniquely human.

TEST YOUR KNOWLEDGE

Episodic Memory Versus Semantic Memory

Episodic memories are memories for the personal experience of autobiographical events, set in a particular time and spatial location; semantic memories are memories for fact or general knowledge about the world, independent of when and how this information was acquired. Sometimes, though, the line between the two is blurred. A single behavior can contain components of both semantic and episodic information. Read the following scenarios to see if you can identify each kind.

([Answers appear at the end of the chapter.](#))

1. A college senior takes his Latin vocabulary exam. The first phrase to be translated is *carpe diem*. This is an easy one; he knows the answer is “seize the day,” even though he cannot remember exactly where he first heard this expression. Is this student using semantic or episodic memory?
2. The second phrase to be translated is *ne tentes, aut perforce*. This is harder; the student can remember studying the phrase, and he even recalls that the phrase was printed in black ink on the lower left of a page in his textbook, but he cannot recall the translation. Is the student using semantic or episodic memory?
3. Later in the day, the senior is helping a new student learn her way around campus. When the tour finishes, the newcomer asks where she can buy a cup of coffee. The senior thinks for a moment, then says that the coffee is better at a nearby Starbucks than at the student center. How might the senior be using both semantic and episodic memory here?

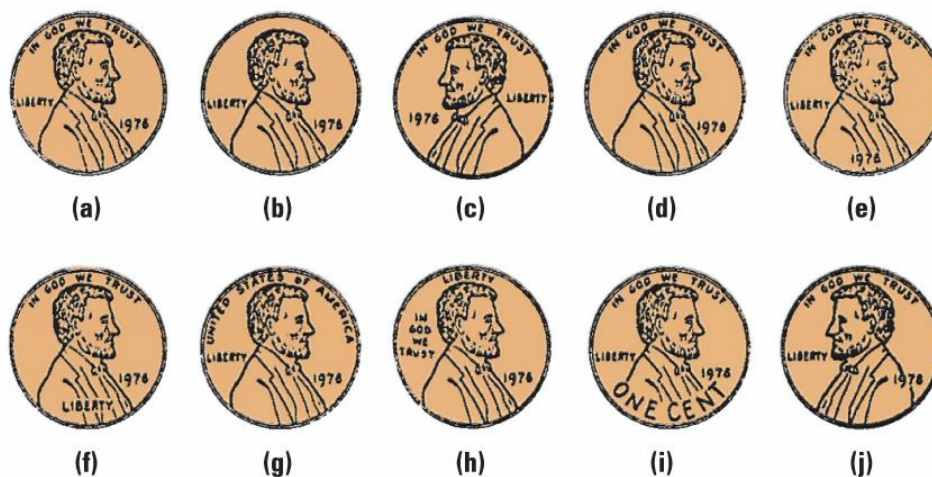
Encoding New Memories

Much of the time, the formation of new episodic and semantic memories seems automatic and effortless. For example, you probably remember a great deal of information about your high school graduation even if you did not spend the day consciously trying to memorize the details so you could recall them later. On the other hand, every student who has ever studied for a test knows that some types of information are easier to learn than others. Here are three basic principles that govern how successfully a new episodic or semantic memory is *encoded*, or stored in memory.

Mere Exposure to Information Does Not Guarantee Memory

On the principle that practice makes perfect, you might think that the best way to learn new information is to study, study, study. But the truth is a little more complicated. Mere repeated exposure to information is not enough to guarantee memory. One telling example of this occurred when BBC Radio in the United Kingdom was planning to change its broadcast frequency. The BBC saturated the airwaves with announcements informing listeners about the new station call numbers. A survey of radio listeners who had heard the announcement at least 25 times a day for many weeks found that fewer than a quarter of these individuals had learned the new call numbers ([Bekerian & Baddeley, 1980](#)). Just presenting the information again and again was not enough to guarantee that listeners would remember.

Memory for visual details can be just as bad. Here is a quick test: try to remember what a U.S. penny looks like. You probably remember that Lincoln's face appears on the front, but can you remember whether he is facing to the left or the right? If you are not sure, you are not alone. Few U.S. students can accurately draw a penny; in fact, few can even pick out an accurate drawing from among a set of fakes ([Nickerson & Adams, 1979](#)). Residents of other countries are just as poor at remembering details of their own national currency ([Jones, 1990](#); [Martin & Jones, 1995](#)). Most of us are quite capable of recognizing coins when we see them. Yet most of us have remarkably poor memory for the details of what these familiar objects actually look like.



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Can you pick out the correct drawing of a penny? Most people have very poor ability to recognize the details of a coin face, though they see and handle the coin every day. What principle of memory does this illustrate?

In short, sheer repetition of either verbal or visual information is not enough to ensure its being remembered. Rather, struggling to learn new information generally promotes better long-term retention of that information, an effect termed the *desirable difficulties* phenomenon ([Soderstrom & Bjork, 2015](#)). You have experienced this for yourself if you have studied hard for an exam, practicing and reviewing the material with attention and effort, and then performed better on the test, compared to another episode where you put less effort into the learning and received a correspondingly lower grade.

Memory Is Better for Information That Relates to Prior Knowledge

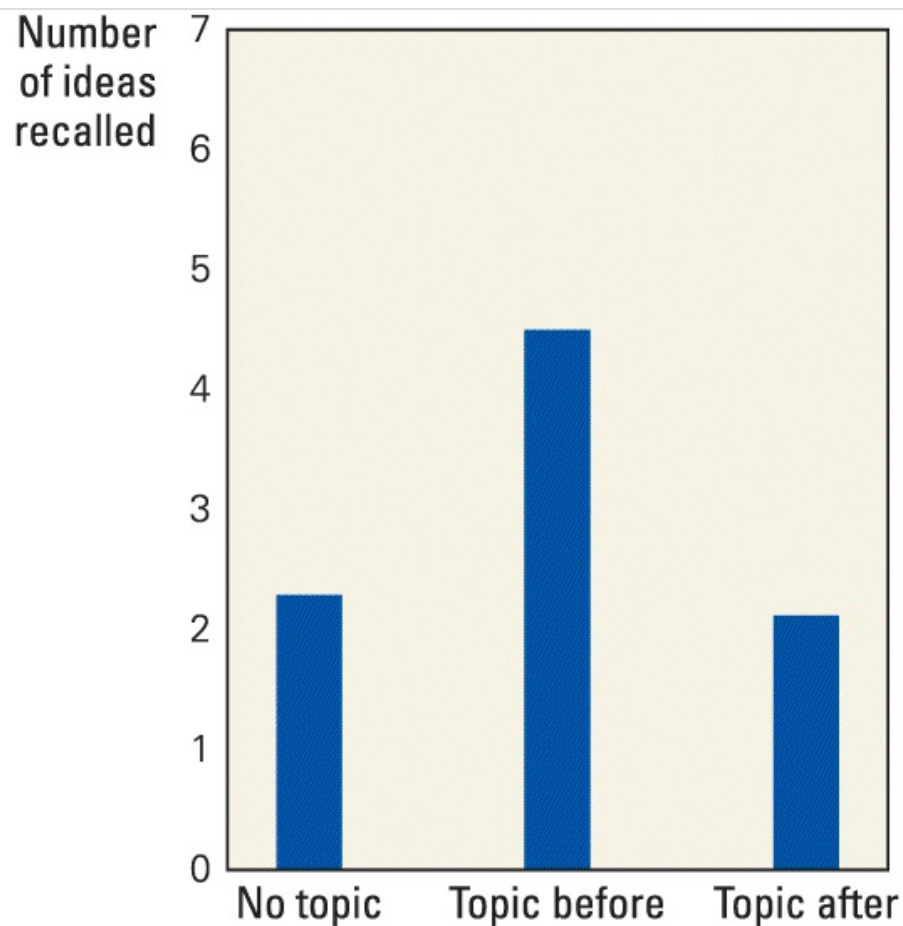
Earlier in the chapter, you read that the Latin word for “arc” or “arch” is *fornix*. To help make this information memorable, we presented the salacious tidbit about Roman prostitutes. The idea was to provide a link between *fornix* and *fornication* (a word you already know), which should help you remember better than if you were just trying to memorize an otherwise meaningless Latin word.

A basic principle of memory is that new information is easier to remember if you can relate it to things you already know. In a classic study, John Bransford and Marcia Johnson read participants a paragraph such as the following:

The procedure is actually quite simple. First, you arrange things into different groups. Of course, one pile may be sufficient depending on how much there is to do. If you have to go somewhere else due to lack of facilities that is the next step, otherwise you are pretty well set. It is important not to overdo things. That is, it is better to do too few things at once than too many.... After the procedure is completed one arranges the materials into different groups again. Then they can be put into their appropriate places. Eventually they will be used once more and the whole cycle will then have to be repeated. However, that is part of life.

([Bransford & Johnson, 1972](#))

Presented in this way, the paragraph makes little sense. Not surprisingly, most participants could recall very little of it later ([Bransford & Johnson, 1972](#)). However, a second group of participants were first told the topic of the paragraph: doing laundry. If you read the paragraph again in light of this background information, you will find that it makes much more sense. Indeed, participants who knew the topic before hearing the paragraph were able to recall twice as much about the paragraph ([Figure 7.4](#)). Importantly, the effect of background information on memory is limited to the process of encoding; it does not help with recall. Thus, people who learned the topic only after they heard the paragraph did not recall the information any better than people who were never told the topic at all. Only people with prior knowledge remembered the paragraph well.



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FIGURE 7.4 The effects of prior knowledge on memory An experimenter read a paragraph aloud to participants. Participants who heard the paragraph by itself (“No topic”) recalled few items, whereas participants who were first informed

of the topic and then heard the paragraph (“Topic before”) recalled significantly more items. Participants who learned the topic only after hearing the paragraph (“Topic after”) performed no better than those who had never heard the topic at all.

Data from [Bransford & Johnson, 1972](#), Table 2.

Description

The Y-axis shows Number of ideas recalled and X-axis shows No Topic, Topic Before, and Topic after. The data in the graph are as follows. No topic: slightly more than 2. Topic before: 5. Topic after: 2.

This principle has clear implications for optimizing study habits. In general, you will remember textbook material better if you take the time to scan a chapter first to get a sense of the major points before reading the details. This is also the reason that many professors encourage students to complete assigned readings before each lecture. With this preparation, students’ minds are better able to encode the information presented in the lecture.

Deeper Processing at Encoding Improves Recall Later

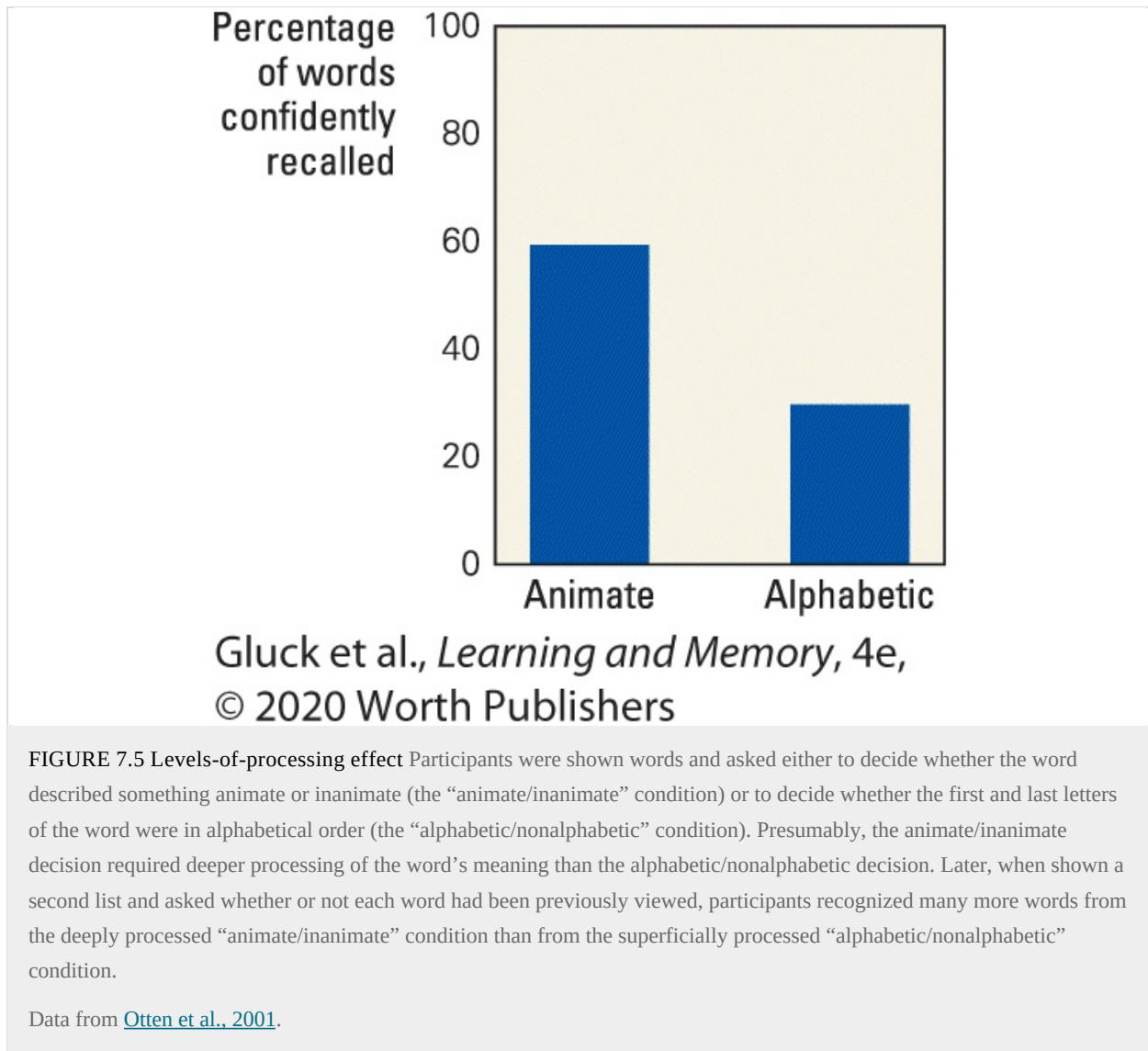
A third general principle is that the more deeply you analyze information, the more likely you are to successfully encode it in memory. Specifically, the [levels-of-processing effect](#) states that the more deeply you process new information during encoding, the more likely you are to recall it later ([Craik & Lockhart, 1972](#); [Craik & Tulving, 1975](#)). If you think about the word *fornix* and its relationship to *fornication*, you are processing the word more deeply than if you just try to memorize the fact that *fornix* = “arch.” Many experiments have shown that people recall words better if they are forced to think about the semantic content (meaning) of the words ([Galli, 2014](#)).

levels-of-processing effect

The finding that, in general, deeper processing (such as thinking about the semantic meaning of a word) leads to better recall of the information than shallow processing (such as thinking about the spelling or pronunciation of a word).

In one such study, participants were shown a list of words presented one at a time. For some of the words, they were told to decide whether the word described something animate or inanimate. For other words, participants were asked to decide whether the first and the last letters of the word were in alphabetical order. Presumably, the “animate/inanimate” decision required thinking deeply about the meaning of the word, but the “alphabetic/nonalphabetic” decision required only superficial thinking about how the letters were arranged. Later, the participants were shown another list and were asked to identify which words they had studied earlier ([Figure 7.5](#)); the deeply processed “animate/inanimate” words were better recognized than the superficially

processed “alphabetic/nonalphabetic” words ([Otten, Henson, & Rugg, 2001](#)).



Description

The Y-axis shows Percentage of words confidently recalled and X-axis shows Animate and Alphabetic. The data in the graph is as follows. Animate: 59%. Alphabetic: 24%.

One criticism of the levels-of-processing idea is that it is vague. How, exactly, can we be sure whether individuals are processing information “deeply” or “superficially”? Just because an experimenter asks participants to think about the spelling of a word, how can we know they are not thinking about the meaning of the word, too? And, for that matter, how can we be sure that

thinking about word meanings requires deeper processing than comparing the first and last letters of a word?

It is hard to answer these questions by using purely behavioral measures, but functional neuroimaging provides some clues. Researchers have used functional magnetic resonance imaging (fMRI) to look at brain activity while participants encode words either “deeply” or “superficially,” and the scans have shown that participants’ brains are much more active during deep-processing trials than during superficial-processing trials ([Davachi, Mitchell, & Wagner, 2003](#); [Otten et al., 2001](#)). This suggests that the brain is indeed working harder during deep processing of information. [Section 7.2](#) identifies specific brain areas that play roles in episodic memory and semantic memory; for now, though, simply note that the psychological concepts of deep versus superficial processing seem to correspond to physiological measures of how hard the brain is working to encode new information.

Retrieving Existing Memories

Of course, successfully encoding a new memory is only half the battle; the other half is successfully accessing, or retrieving, that memory when we want it. You have probably experienced the “tip-of-the-tongue” phenomenon, when you’ve tried to summon a word or a name from memory that you were sure you knew but simply could not retrieve at the moment. In these cases, the information was not permanently lost; it was only temporarily inaccessible. You may have succeeded in recalling the information later, after you had turned your attention to something else.

Why can we retrieve stored memories at some times, yet at other times they elude us? Like encoding, successful retrieving is governed by several basic principles. Here are three key principles that determine how successfully memory is retrieved.

Encoding Specificity: Similar Conditions at Encoding and Retrieval

One factor in our ability to retrieve a memory is how closely the current context resembles the context in which the information was originally acquired. The [encoding specificity effect](#) states that retrieval is more likely to be successful if the conditions at recall are similar to those that

occurred at encoding ([Tulving & Thompson, 1973](#)).

encoding specificity effect

The principle that retrieval is likely to be more successful if the cues and contextual conditions present at recall are similar to those that were present at encoding.

For example, suppose you are initially shown a series of pictures of objects (a dog, a house, and so on) and are later given a recognition test in which some of the objects are presented as pictures and some as words. Which objects do you think you would remember best? Most people show better recognition if the format is the same at encoding as at testing: if objects presented as words are tested as words, and objects presented as pictures are tested as pictures ([Köhler, Moscovitch, Winocur, & McIntosh, 2000](#)). Performance is worse when the encoding and testing formats differ.

The encoding specificity principle also applies to the context or physical location where learning takes place. Have you ever been at the gym or the supermarket and run into someone you know from school and been temporarily unable to recognize that person in the unusual setting? You may even have struggled to chat for a while (without admitting you could not remember exactly who this person was) before something “clicked” and the memory fell into place. If you have ever had this type of experience, then you already know the powerful effect of physical context on memory retrieval.

In a famous demonstration of this principle, researchers tested memory in members of a diving club ([Godden & Baddeley, 1975](#)). Some of the divers were asked to learn a list of 40 words while on dry land; the remainder learned the list underwater. The divers were then tested on their recall of the words. Divers who were tested in the same environment where they had studied the list (either on land or underwater) could remember more words than those who were trained in one environment but tested in the other. Similarly, students who learn a list either standing up or sitting down will later recall a few more words if they are in the same position during testing ([Godden & Baddeley, 1975](#)). A list learned while classical or jazz music plays will be remembered better if the same music plays during recall ([Smith, 1985](#)). In each case, recall is slightly better if the retrieval conditions are similar to the encoding conditions.

So does this mean that studying in the same room where you will take a test will improve your performance? Not necessarily. A large study of 5,000 college students found no effect on

performance whether final exams were administered in the same room where the course had been taught or in a novel classroom ([Saufley, Otaka, & Bavaresco, 1985](#)). This may be because most classrooms are pretty much alike; however, if your professor tells you that the exam will take place underwater, you should probably prepare accordingly.

Transfer-Appropriate Processing: Similar Processing at Encoding and Recall

The encoding specificity principle is about similarity in format (e.g., visual versus auditory) and context (e.g., classroom versus underwater) between encoding and retrieval. A related principle, the **transfer-appropriate processing (TAP) effect**, states that memory will be best when the way in which information is processed at encoding matches the way it is processed at retrieval ([Morris, Bransford, & Franks, 1977](#)). In other words, it is better to study (“process”) material in the same way that it will be tested.

transfer-appropriate processing (TAP) effect

The principle that memory retrieval will be best when the way that information is processed at encoding matches how it will be processed at retrieval.

For example, remember the levels-of-processing effect, in which “deeper” processing leads to better memory than “superficial” processing? This is really a transfer-appropriate processing effect in disguise. People who process a word “deeply,” thinking about its meaning and visualizing it, are indeed better at a standard visual recognition test (as you saw in [Figure 7.5](#)). But people who are asked to merely rhyme a word—a “superficial” processing task that does not involve thinking about the word’s semantic meaning—actually perform better if the later memory test involves rhyming recognition ([Morris et al., 1977](#)). In short, deep processing during encoding is best if the test also requires deep processing; but if the test instead involves the physical attributes or sounds of a word, superficial processing during learning may be preferable!

You can use the principle of transfer-appropriate processing to your advantage while studying. For instance, suppose you can study for an exam either by taking online multiple-choice tests or by recruiting a friend to ask you open-ended questions from your class notes. If you know that the professor usually gives essay exams, which study method should you use? The best way to prepare for a test is by processing the material in a way that is similar to how you expect to be tested on it, making the study and recall formats as similar as possible ([Butler & Roediger,](#)

2007).



"THEN, AS YOU CAN SEE, WE GIVE THEM
SOME MULTIPLE CHOICE TESTS."

Sidney Harris/ScienceCartoonsPlus.com

Even if rats could take multiple-choice tests, they would probably perform very poorly on this one. What principle of strong memory retrieval does this multiple-choice test violate?

More Cues Mean Better Recall

Of course, many formats are available for testing memory. The most obvious is **free recall**, in which you are simply asked an open-ended question and you supply the answer from memory (What is the Latin word for “arch”?). A second possibility is **cued recall**, in which you are given some kind of a prompt or clue to elicit the correct answer (What is the Latin word for “arch”? F___). A third possibility is **recognition**, in which you pick out the correct answer from a list of possible options (What is the Latin word for “arch”? A = *fenestra*, B = *fornix*, or C = *fundus*).

free recall

A memory test that involves simply generating requested information from memory.

cued recall

A memory test that involves some kind of prompt or cue to aid recall.

recognition

A memory test that involves picking out (or recognizing) a studied item from a set of options.

In general, free recall is harder than cued recall, which in turn is harder than recognition. This ranking directly reflects the number of cues available to jog the memory. In free recall, the tester provides no (or few) explicit cues; in cued recall, the tester provides at least some kind of cue; and in recognition, the entire item is provided. In one study, when asked to recall the names of their high school classmates, recent graduates could, on average, produce about 50% of the names; individuals who had graduated several decades earlier could produce only about 20 to 30% of the names. But when shown a list of names and asked to recognize which people had been classmates, recent graduates could get about 90% correct, and even long-ago graduates got about 85% correct ([Bahrick, Bahrick, & Wittlinger, 1975](#)).

Most people instinctively understand that free recall is more difficult than recognition. This is one reason many students prefer exams that contain multiple-choice rather than essay questions (recognition as opposed to free recall). Of course, professors understand this, too, and they usually compensate by designing multiple-choice questions to include choices that can easily be mistaken for the correct response if a student has not studied the material closely. (Have you ever wished you were better at memorization? See [“Learning and Memory in Everyday Life”](#).)

LEARNING AND MEMORY IN EVERYDAY LIFE

Extraordinary Memorizers

At the 2017 International Association of Memory (IAM) World Championship, contestants memorized information—such as hundreds of digits, or the order of a deck of cards—as quickly as possible. Champion Alex Mullen, an American medical student, memorized 568 numbers in 5 minutes.

“I definitely didn’t [always] have a great natural memory,” Mullen says, “but in 2012 I started training” ([Thomson, 2016](#)). Most memory champions use strategies called *mnemonics* (pronounced “nee-MON-ics”), which are techniques that make information easier to memorize. Like many memory champions, Mullen uses the “method of loci” (also called “mind palace”), a mnemonic technique that dates back at least to the time of the ancient Greeks. Using this method, you visualize a familiar place, such as your home, and imagine walking through it, leaving items to be remembered at specific points along the way. For example, to memorize the order of a shuffled deck of playing cards, if you are a well-prepared “memory athlete,” you might first have assigned an image to each card: a cup for the seven of diamonds, a guitar for the three of spades, and so on. You then walk through your house, visualizing a cup of tea at the front door, a guitar further along the entrance hallway, and so on along a predetermined path. To recall the items later, you mentally retrace the route, recalling

each item in turn. Memory champions typically practice such strategies several hours a day, every day, until the routines become almost effortless—and fast enough to win championships.

The IAM organizers consider memory contests a sport in which—as in any sport—the top athletes increase their skill through hard practice. In fact, memory athletes do not typically score above average on tests of general cognition or intelligence ([Maguire, Valentine, Wilding, & Kapur, 2003](#)). Unsurprisingly, on memory tests that involve lists of words or numbers (like many of the events in memory championships), the memory athletes perform very well; but when asked to study and recall other types of information, such as visual images, they typically score no better than average. Structural MRI shows no obvious abnormalities in the brains of the memory athletes, but functional MRI of the athletes shows heightened activity in some brain areas, such as the hippocampus, while they are memorizing new information—consistent with the idea that these are “normal” people who have learned to use special strategies to encode new information.

So can anyone learn to be a memory athlete? In one study, a group of volunteers with normal memory skills, given 6 weeks’ training in mnemonic techniques, showed significant improvement on laboratory tests of memory, and their brain activity patterns began to look more like those of the memory athletes ([Dressler et al., 2017](#)). The implication is that almost anyone could become a memory athlete by investing the time to master a mnemonic system ([Ericsson, 2003](#)).

And what about in real life? Memory athletes claim that they successfully apply their mnemonic techniques for daily chores, such as remembering a shopping list or even—in Mullen’s case—studying for an exam. But they also confess that they have the same day-to-day memory challenges as the rest of us, such as forgetting where they left their car keys or where they parked the car ([Handwerk, 2017](#)).

Struggling (and Even Failing) to Remember Can Improve Memory

Students and teachers usually think of studying (encoding) as being distinct from testing (retrieval), with the latter mainly serving to assess whether learning was successful. But, in fact, the mere act of taking a test can serve as a powerful enhancer of later memory for the information tested. This “testing effect” on memory has been observed numerous times in many conditions (for review, see [Roediger & Butler, 2011](#)). In fact, a difficult test—one that forces you to struggle to retrieve the information—may be most helpful of all. These benefits can occur even if you fail to remember the material during the test and even if you do not receive feedback on whether your answers were correct.

In one experiment, students were asked to study two short paragraphs. Later, they were asked to reread one of the paragraphs (the “reading condition”); for the other paragraph, they were given a blank sheet with the title printed at the top and were asked to write down all the material they could remember (the “testing condition”). A week later, students recalled only about 40% of the ideas from the paragraph they had reread—but they successfully recalled about 55% of the ideas

from the paragraph they had previously been tested on ([Roediger & Karpicke, 2006](#)). Thus, prior testing improved later recall—even though no feedback had been provided during the test.

The “testing effect” may be another example of the *desirable difficulties* phenomenon, in which struggling to recall information can actually promote retention of that information later. In fact, several studies suggest that if instructors periodically interrupt a lecture to give short quizzes, perhaps polling students via electronic “clicker” devices, students perform better when challenged to retrieve information from that lecture on a subsequent exam (for review, see [Glass & Sinha, 2013](#)).

The next time you have to study for a test, remember these principles: instead of just rereading the chapter, consider taking an online quiz (if you think the exam you are studying for will include multiple-choice questions), using flashcards (if the exam will require short answers), or starting with a blank piece of paper and writing down everything you can remember about the topic (if you think the exam will include essay questions). Chances are, the effort you put into recalling the information during your practice test will pay off later, on the actual exam.

When Memory Fails

You have just read about some basic principles that affect encoding and retrieval of episodic and semantic memory. At each stage, various conditions (format, context, etc.) can help or hinder the process. Considering all the opportunities for failure, it is amazing how often our memories serve us well.

But there are many situations in which episodic memory and semantic memory do fail us. The tip-of-the-tongue phenomenon, mentioned earlier, is an example of a temporary failure: the memory is inaccessible at the moment but may resurface later. In other cases, though, memories are truly lost or corrupted. Many failures of memory reflect one of four basic phenomena: simple forgetting, interference from other memories, source monitoring errors, and false memory. We will discuss these in more detail next.

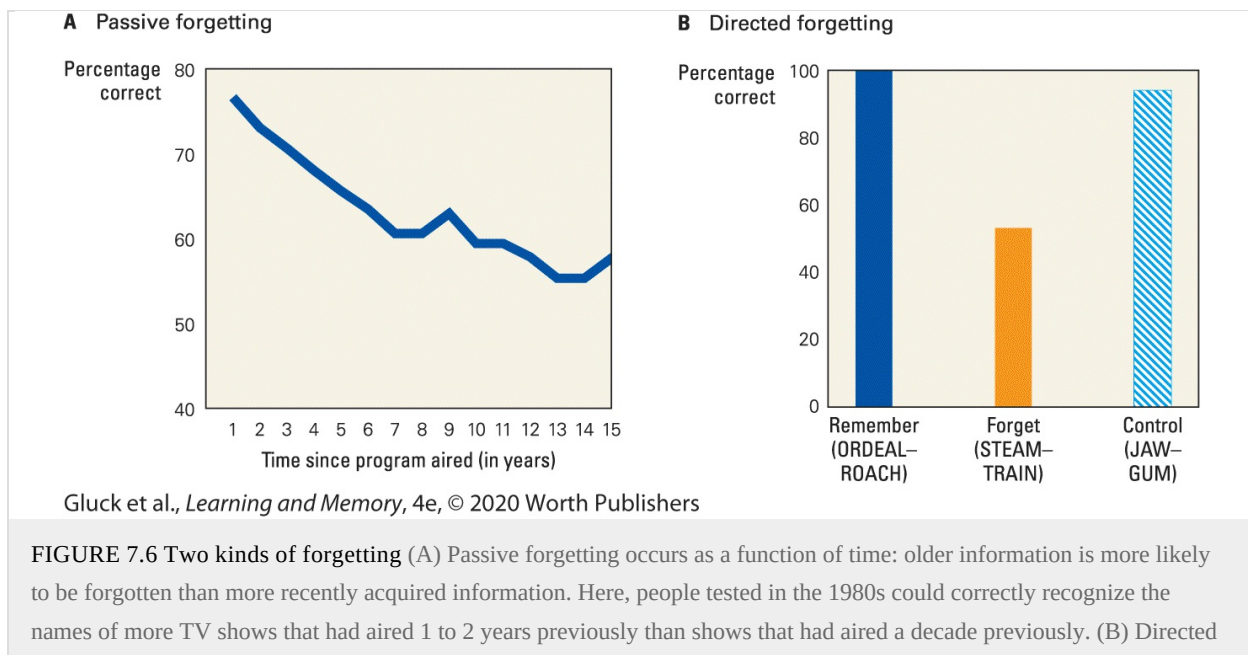
Forgetting

It is probably obvious that you are more likely to remember things that happened recently than things that happened long ago. For example, you probably remember most of the events that

have happened to you today but not what happened to you on, say, July 16, 2017. However, if someone had questioned you on July 17, 2017, you probably could have reported the prior day's events in great detail. Somehow, that information has trickled away as the months passed. What governs how fast we forget?

[Chapter 1](#) describes how Hermann Ebbinghaus conducted a series of early studies to quantify human learning and forgetting. Specifically, he memorized lists of nonsense words and then tested his own memory for the items. He concluded that most forgetting occurs in the first few hours or days after learning ([Ebbinghaus, 1885/1964](#)). Information that survives the critical first few days might last in memory indefinitely.

Ebbinghaus's basic finding has since been replicated in a variety of studies. For instance, in the 1980s, memory researcher Larry Squire developed a test in which people were queried about television shows that had aired for a single season from 1 to 15 years earlier ([Squire, 1989](#)). (In those days, there were only a handful of television channels, and most people were familiar with the names of most programs that aired.) On average, people did quite well on this test of semantic memory. Most people could correctly recognize the names of more than 75% of TV shows that had aired in the prior year, although they recognized progressively fewer shows from earlier years ([Figure 7.6A](#)). Most forgetting occurred within the first decade, so people remembered almost as many TV shows from 15 years before as from 10 years before.



forgetting occurs when we intentionally try to suppress memory. Here, participants' memory was worse for studied word pairs they had been instructed to forget than for pairs they had been instructed to remember or control pairs they had not seen since the original study phase.

(A) Information from [Squire, 1989](#), Figure 1. (B) Information from [Anderson et al., 2004](#), Figure 1B.

Description

The X-axis shows Time since program aired (in years) and Y-axis shows Percentage correct. The data in the graph are as follows. The line graph shows a steady decrease in its value till 7 years. Then, the line continues to decrease, but with greater variation.

The Y-axis shows Percentage correct, and X-axis shows Remember (ORDEAL–ROACH), Forget (STEAM–TRAIN), Control (JAW–GUM). The data in the bar graph is as follows. Remember (Ordeal and Roach): 100%. Forget (Steam and Train): 55%. Control (Jaw and Gum): 95%.

We normally think of forgetting as a passive process in which old memories—particularly those we have not accessed in a while—just quietly fade away. But we may have more power over what we remember (and forget) than we realize. **Directed forgetting** occurs when information is forgotten on demand. For example, Michael Anderson and colleagues trained participants on a series of word pairs: ORDEAL–ROACH, STEAM–TRAIN, JAW–GUM, and so on ([Anderson et al., 2004](#)). Next, the researchers showed participants some of the studied words. For some words (ORDEAL), people were now asked to *remember* the associated word (ROACH). For others (STEAM), they were asked to try to *forget* the associated word (TRAIN). Later, Anderson tested memory for all the studied words ([Figure 7.6B](#)). People were less able to remember the words they had tried to forget than those they had tried to remember or those they had not seen since the original training (GUM). Note that even in the “forget” condition of [Figure 7.6B](#), people still remembered about 50% of the items, on average.

directed forgetting

A procedure in which participants are first asked to learn information and later asked to remember or forget specific items; typically, memory is worse for items that a participant was directed to forget.

Thus, directed forgetting is not perfect. But these results suggest that we have some control over what we do and do not forget. Other studies have even shown that intentional forgetting can extend to autobiographical events: participants who kept a daily diary and were then instructed to forget the previous week's events subsequently recalled fewer events than participants who kept a diary but did not receive instructions to forget ([Joslyn & Oakes, 2005](#)).

Obviously, intentional forgetting in the laboratory may be very different from forgetting in real life. Nevertheless, the fact that it is theoretically possible to intentionally forget unwanted information may help explain some real-world cases of memory suppression in which individuals forget highly traumatic and unpleasant events such as war, rape, or natural disasters. And it may be possible to exploit this ability in developing therapy for psychiatric disorders in which people suffer from intrusive and unwanted thoughts, such as posttraumatic stress disorder and obsessive compulsive disorder. In this way, the “memory failure” of forgetting may actually have some psychological benefits.

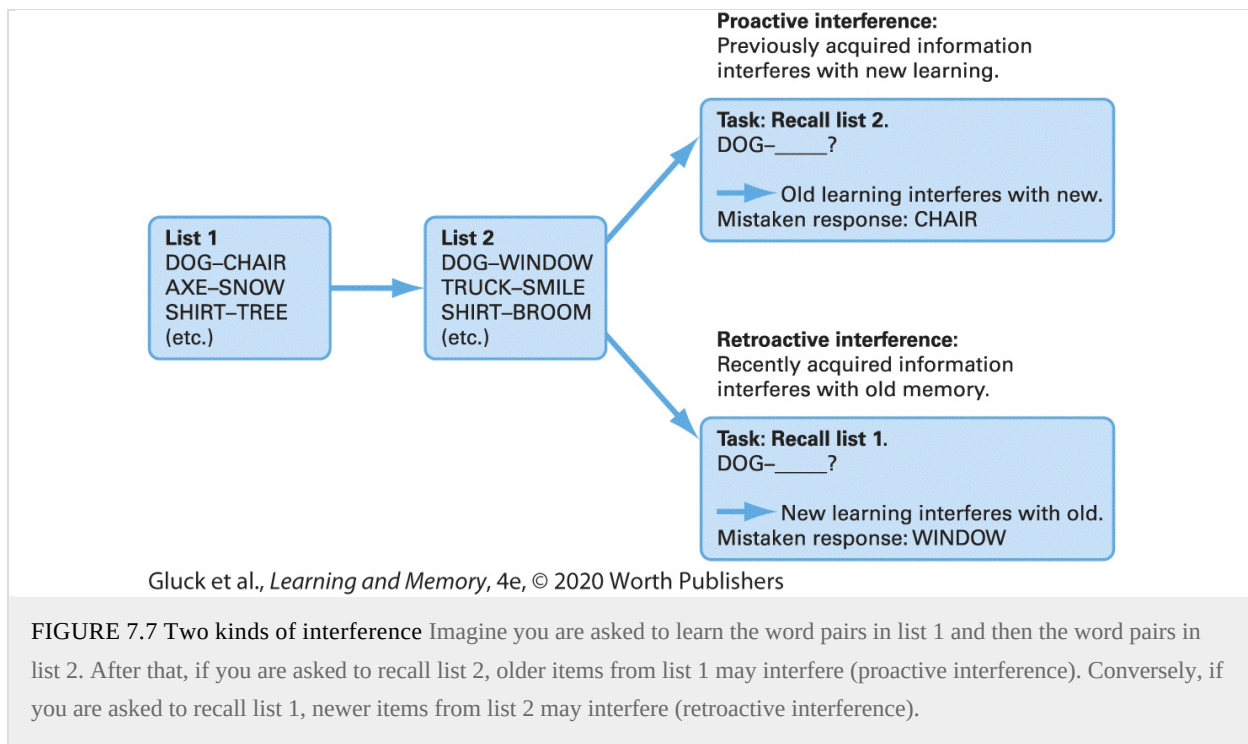
Interference

Remember the parking lot scenario, in which memories of prior days’ parking locations disrupt your ability to recall where you parked the car today? This is an example of **interference**, in which the strength of a memory may be reduced due to overlap with the content of other memories.

interference

Reduction in the strength of a memory due to overlap with the content of other memories.

Suppose you are participating in a memory experiment, and the experimenter asks you to learn a list of word pairs—say, list 1 in **Figure 7.7**. You might practice this list, repeating it aloud, until you have it memorized. Then, after some delay, the experimenter gives you a test of cued recall: given the stems (e.g., DOG—_____), fill in the appropriate associate (CHAIR) for each stem.



Description

The flow begins with a text box, List 1 DOG-CHAIR AXE-SNOW SHIRT-TREE (etc.). It leads to a text box, List 2 DOG-WINDOW TRUCK-SMILE SHIRT-BROOM (etc.). It branches into two text boxes which read, Task: Recall list 2. DOG-____? Old learning interferes with new Mistaken response: CHAIR and Task: Recall list 1. DOG-____? New learning interferes with old mistaken response: WINDOW. The corresponding text boxes read, Proactive interference: Previously acquired information interferes with new learning and Retroactive interference: Recently acquired information interferes with old memory, respectively.

Now suppose the experimenter asks you to memorize a second list, list 2 in [Figure 7.7](#). Note that some of the items (DOG, SHIRT) appear in both lists. As you attempt to learn the new pair DOG-WINDOW, the stem (DOG) will stimulate recall of the old associate (CHAIR). This may interfere with your learning of list 2, and when it comes time to test your memory for list 2, you may mistakenly respond with the old associate (CHAIR) instead of the new one (WINDOW). This process, whereby old information can disrupt new learning, is called **proactive interference** ([Anderson, 1981](#); [Wickelgren, 1966](#)).

proactive interference

Disruption of new learning by previously stored information.

The opposite process also occurs: suppose that, after heavy practice with list 2, you try to go

back and recall list 1. Now, when the experimenter prompts DOG—_____, you might recall WINDOW from list 2 instead of CHAIR from list 1. This process, whereby new information can disrupt old learning, is called **retroactive interference**. A simple mnemonic to help you remember the difference between proactive interference and retroactive interference is that *PR*oactive interference means *PRE*viously acquired information is at fault; *RE*troactive interference means *RE*cently acquired information is at fault.

retroactive interference

Disruption of old (previously stored) information by more recent learning.

Proactive and retroactive interference occur in many real-life contexts. For example, the last time you changed a frequently used computer password, you probably went through a phase during which you sometimes typed the old password by mistake. This is an example of *proactive* interference, where memory of the *previous* password interferes with your ability to retrieve memory for the new one. On the other hand, once you have successfully mastered a new password, you might have some trouble remembering old passwords you once used. This is an example of *retroactive* interference, as memory of the *recently* acquired password interferes with your ability to remember the old ones.

Source Monitoring

Some memory failures are **source monitoring errors**, which occur when we remember information but are mistaken about the specific episode that is the source of that memory. For instance, while waiting in line at the supermarket, we may read some gossip on the cover of a trashy tabloid, and even though we know better than to take such reading material seriously, we may later remember the gossip but forget the dubious source—and thus give the rumor more credence than it deserves. In effect, the semantic content of the memory is preserved, but the episodic details (the time and place in which we encountered the information) are distorted.

source monitoring error

Remembering information but being mistaken about the specific episode that is the source of that memory.

A famous real-world example of an apparent source monitoring error occurred in the early 1970s, when ex-Beatle George Harrison released the hit single “My Sweet Lord” and was sued for copyright infringement because the tune was so similar to an earlier song, the Chiffons’ “He’s So Fine.” Harrison argued that “My Sweet Lord” was his own creation and denied any

plagiarism, although he admitted to having heard the Chiffons' song. A judge ruled that Harrison had indeed been influenced by his memories of the earlier song, although the judge was convinced that Harrison's plagiarism was unintentional. Apparently, the ex-Beatle had suffered a type of source monitoring error: remembering the melody and mistakenly thinking he had composed it himself. Numerous musicians have faced similar charges, and many have similarly claimed the plagiarism was unintentional—even as they paid fines or settled out of court.

Similarly, source monitoring errors can occur when a student reads a sentence in a textbook and remembers it; later, when the sentence pops to mind, it “feels” new and gets included in a term paper. The student may honestly think this is an original idea—but if the instructor has read the same textbook, accusations of plagiarism will follow.

False Memory

Related to the phenomenon of source monitoring errors is [false memory](#), or memory for events that never actually happened. Elizabeth Loftus and her colleagues have conducted several studies in which they intentionally “implanted” false memories of childhood events in ordinary people. For instance, in one study, the researchers invented several fictitious events, such as getting lost in a shopping mall or surviving a vicious animal attack. An experimenter then told research participants that these events had happened to the participants when they were children ([Loftus & Pickrell, 1995](#)). Family members, who had agreed to collaborate, also spoke about the events as if they had really happened. Sure enough, a few days later, about 25% of the participants seemed to believe the events were real—and even “remembered” additional details that had not been present in the original story.

false memory

Memory for events that never actually happened.

In another study, Kimberley Wade and her colleagues pasted childhood photos of their adult research participants into a photograph of a hot-air balloon ride. The researchers then showed participants the doctored photos and asked them to describe everything they could remember about the fictitious ride. After three such sessions, about half the people in the study claimed to remember having taken the ride—even though none had ever been in a hot-air balloon ([Wade, Garry, Read, & Lindsay, 2002](#)).

In laboratory research, false memories are particularly likely to occur when people are prompted to imagine missing details; later, they may mistakenly remember those details as the truth. The more that people imagine an event, the more likely they are to subsequently believe it really happened ([Goff & Roediger, 1998](#); [Thomas & Loftus, 2002](#)).

Outside the lab, false memory may be just as common. The online political magazine *Slate* conducted a mass experiment in altering readers' memories. *Slate* showed readers photos of several highly controversial political events, such as Florida Secretary of State Katherine Harris presiding over the hotly disputed 2000 presidential election recount. Intermixed with these pictures were photos of events that never actually happened, such as President Obama shaking hands with Iranian President Ahmadinejad, or President G. W. Bush relaxing at his ranch while Hurricane Katrina was devastating New Orleans. The fake photos had been constructed by altering real pictures, as in [Figure 7.8](#), which shows how an existing photo of Obama was doctored to include Ahmadinejad. Readers visiting the *Slate* website were asked whether they remembered each picture. More than 5,000 readers responded. While most people reported remembering the real photos, almost half said they also “remembered” the fictitious events, and many provided comments describing how they had first heard the “news” depicted in the false photos ([Frenda, Knowles, Saletan, & Loftus, 2013](#)). These results are particularly striking because visitors to the *Slate* website were presumably interested in and well informed about political events. Yet even they fell victim to the false-memory manipulation.



Photo illustration by Slate, original images by Getty; left: SAUL LOEB/AFP/Getty Images, right: JUAN BARRETO/AFP/Getty Images.

FIGURE 7.8 Creating false memories in the real world The online political magazine *Slate* displayed photos of controversial political events and asked readers if they remembered the photos and the events they depicted. In reality, several of the pictures represented events that never occurred; for example, the image here of President Obama shaking

hands (A) was doctored to include the president of Iran (B). Approximately half of *Slate*'s politically savvy respondents "remembered" the fictitious events.

The apparent pervasiveness of false memories should be a matter of public concern, particularly in criminal court cases where the strongest evidence against a suspect is eyewitness testimony. Loftus and other false-memory researchers have been vocal in warning that the types of procedures that induce false memory in the lab must be scrupulously avoided in the justice system ([Loftus, 1996, 2003](#); [Radelet, 2002](#); [Wells, Memon, & Penrod, 2006](#)). For example, if an eyewitness is shown the photo of a suspect, the witness's memory of the photo may become confused with the memory of the actual crime, leading the witness to "recognize" the suspect as the perpetrator even though the suspect is innocent. Examples abound to suggest that such mistakes occur all too frequently. One study reviewed 62 cases in which people were convicted of crimes and later exonerated based on DNA evidence ([Neufield & Dwyer, 2000](#)). In more than 80% of these cases, the crucial evidence leading to conviction was eyewitness testimony in which witnesses had mistakenly identified people later proven to be innocent.

Even firsthand confessions may sometimes be unreliable. Laboratory studies suggest that with appropriate prompting, a majority of research participants can be led to develop false memories of committing a crime and to volunteer a detailed false account of that incident ([Shaw & Porter, 2015](#)). This creates concerns about real-world convictions based on confession evidence, particularly if the confession was obtained using interrogation tactics that can encourage creation of false memories. In fact, false confessions play a role in about 25% of wrongful convictions later overturned by DNA evidence ([Kassin, Bogart, & Kerner, 2012](#)). Some of these individuals pled guilty to avoid risk of a harsher sentence, but some may have been misled by their own false memories of having participated in the crime.



Photographiee.eu/Shutterstock

How might showing an eyewitness a photo of the prime suspect and saying “We think this is the guy” influence the witness’s memory of the crime scene?

This does not mean that eyewitnesses—and even confessions—are inherently unreliable. In fact, two field studies comparing eyewitness descriptions of crimes to recordings of the same crimes captured on closed-circuit TV found the eyewitness descriptions to be about 90% correct ([Wixted, Mickes, & Fisher, 2018](#)). But it is critically important that law enforcement personnel avoid using techniques that are likely to promote false memories, such as showing witnesses a photo of someone they describe as a prime suspect or suggesting that witnesses “try harder” if they are unsure of what they recall ([Loftus, 1996](#); [Wells et al., 2006](#); [Wixted et al., 2018](#)). In October 2014, the National Academy of Sciences published a report outlining proposed reforms to eyewitness procedures, many based on findings from false-memory research; as of 2018, 19 states had implemented some or all of these policies in their justice systems ([Innocence Project, 2018](#)).

Memory Consolidation and Reconsolidation

Passive-forgetting curves, such as the one shown in [Figure 7.6A](#), suggest that if you can still remember a fact or an event after a few months, then the odds are good that you will remember it permanently (or at least for a very long time). One implication of this finding is that semantic

and episodic memories are subject to a [consolidation period](#), a time window during which new memories are vulnerable and easily lost ([Dudai, 2004](#); [McGaugh, 2000](#); [Ribot, 1882](#)).

consolidation period

A length of time during which new episodic and semantic memories are vulnerable and easily lost or altered; each time a memory is recalled, it may become vulnerable again until it has been “reconsolidated.”

The Consolidation Period

In an early demonstration of consolidation periods, Carl Duncan trained rats to make a simple operant response (the kind of learning described in [Chapter 5](#)). He then gave the rats [electroconvulsive shock](#), a brief pulse of electricity delivered to the brain via electrodes on each side of the head. If the shock was given 20 seconds after the end of training, the rats’ conditioned response (i.e., the “memory” of the association they had learned) was severely disrupted. However, if the shock was given an hour or more after training, there was little disruption. Intermediate delays led to intermediate levels of disruption ([Duncan, 1949](#)). Thus, the consolidation period for this type of learning in rats appears to extend for a few minutes; older memories (from several hours before) are relatively stable and difficult to disrupt, but more recent memories (from less than a minute before) are highly vulnerable to disruption.

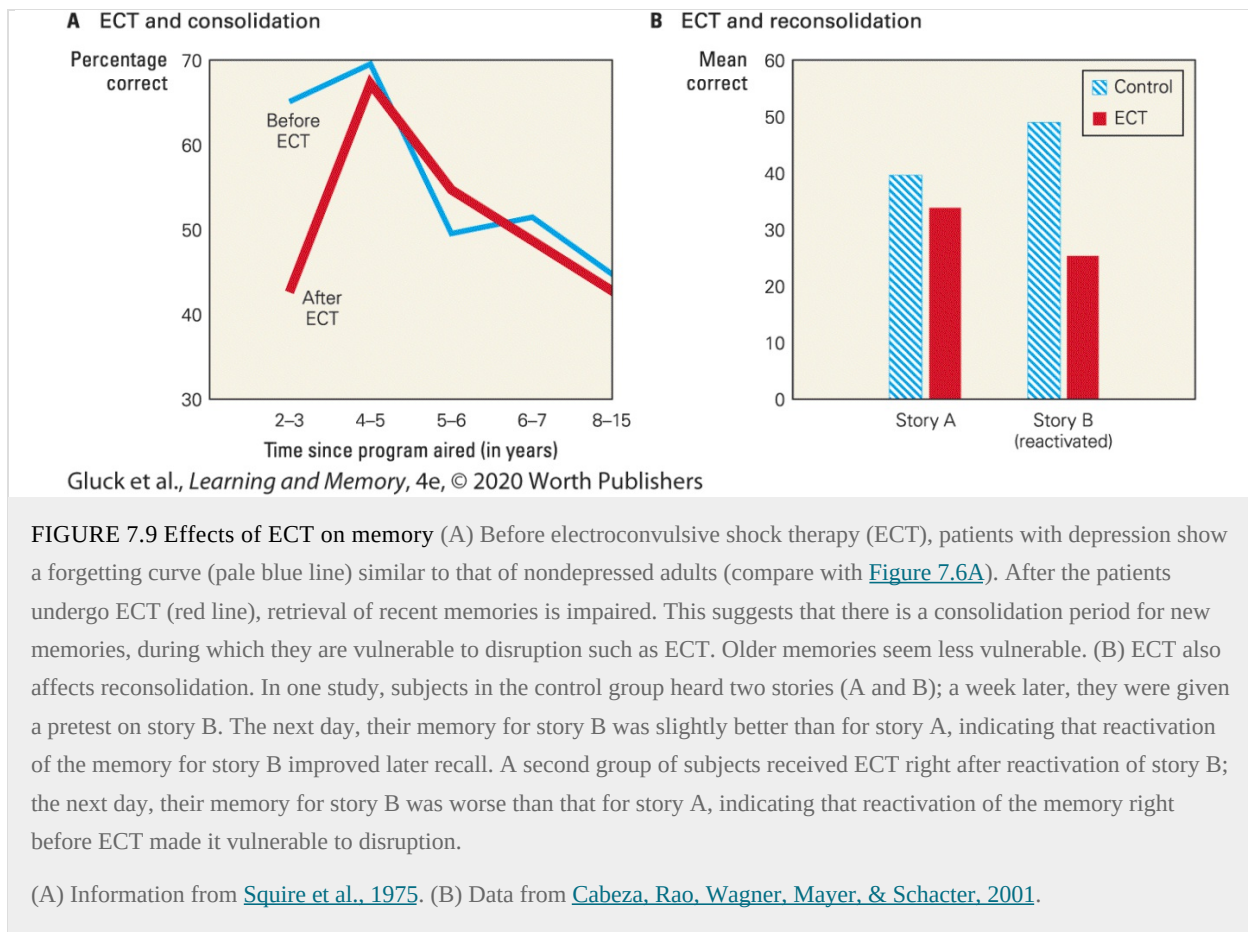
electroconvulsive shock

A brief pulse of electricity that is passed through the brain and can severely disrupt newly formed memories.

Electroconvulsive shock is sometimes administered to humans to provide temporary relief from certain kinds of mental illness, particularly in severe depression. Patients are given general anesthesia and a muscle relaxant beforehand. No one knows exactly why the procedure, called *electroconvulsive therapy*, or *ECT*, relieves depression, but patients often experience relief for weeks or even months afterward ([Glass, 2001](#); [National Institutes of Health Consensus Conference, 1985](#)).

By studying patients who are undergoing ECT, researchers have been able to investigate the effects of electroconvulsive shock on human memory. For example, Larry Squire and his colleagues administered the TV-show test to patients with severe depression ([Squire, Slater, & Chace, 1975](#); [Squire, Slater, & Miller, 1981](#)). Before an ECT session, the patients remembered recent shows (from 2 to 3 years earlier) very well and older shows (from 8 to 15 years earlier) less well ([Figure 7.9A](#), pale blue line). This is similar to the performance of healthy adults,

which you saw in [Figure 7.6A](#). A week after ECT, the same patients retook the TV-show test. Almost invariably, they had forgotten some of the information they had reported earlier, especially shows that had aired within the past 2 to 3 years; older memories (4 to 15 years) were generally unaffected ([Figure 7.9A](#), red line). A similar pattern was seen for episodic memories of autobiographical events ([Squire, Cohen, & Zouzonis, 1984](#); [Vakil et al., 2000](#)). For many patients who undergo ECT, this limited memory loss is a small price to pay for relief from the debilitation of severe depression.



Description

The graph on the left is labeled ECT and consolidation. The X-axis shows Time since program aired (in years) and Y-axis shows Percentage correct. The data in the graph is as follows. The graph shows two lines: Before ECT and After ECT. The line Before ECT shows recent memories were easily accessed (65 to 70% correct). The line After ECT shows recent memories was not retrievable (45% correct).

The graph on the right labeled ECT and reconsolidation. The X-axis shows Story A, and Story B (reactivated) and Y-axis shows Mean correct. The data in the graph shows: Story A (not reactivated) shows values close to 35 for both control and ECT group. Story B (reactivated) showed values of 25 for ECT group and 50 for the control group.

Reconsolidation

The idea of a finite consolidation period, during which new memories are vulnerable to disruption, is attractive because it accounts for a great deal of data from animals and humans. Recently, though, new data is resurrecting an old idea that no memory, however ancient, is really permanent and immutable ([Lee, Nader, & Schiller, 2017](#); [Nadel & Land, 2000](#)). For example, it has long been known that if an old memory is recalled just before electroconvulsive shock is administered, it can be disrupted—just like newer memories (e.g., [Misanin, Miller, & Lewis, 1968](#)). Apparently, each time an old (presumably consolidated) memory is recalled or reactivated, it may become vulnerable again, in a process termed **reconsolidation**.

reconsolidation

The process whereby each time an old memory is recalled or reactivated, it may become vulnerable to modification.

In one study of reconsolidation ([Kroes et al., 2014](#)), two groups of patients undergoing ECT to treat depression were told two stories (A and B), each accompanied by a slide show. A week later, some patients (the control group) were given a short pretest on story B. As you read above, the mere act of testing can improve memory, so you should not be surprised to learn that when given a final multiple-choice test on both stories the next day, the control group remembered more of story B than story A ([Figure 7.9B](#)). As a matter of fact, the testing effect itself may be an example of reconsolidation: reactivating story B allowed the memory of it to become modifiable, which in this case strengthened the memory.

But a second group of patients (the “ECT group”) was given the same pretest on story B, followed immediately by ECT. The next day, on the multiple-choice test, this group’s memory for story A was about as good as the control group’s—but its memory for story B was much worse. In other words, reactivating the memory for story B right before ECT made it vulnerable to disruption by the ECT. Story A, which had not been reactivated, was safe. Memories can also be disrupted if they are reactivated just before administration of drugs that block the formation or maintenance of synaptic connections ([Nader, Schafe, & LeDoux, 2000](#); [Przybylski & Sara, 1997](#)).

The opposite effect can be seen after brain stimulation. For example, [Chapter 2 \(Figure 2.15\)](#)

describes *transcranial direct current stimulation* (tDCS), a process in which experimenters apply weak electrical current to the scalp, temporarily increasing excitability of the neurons below. When tDCS is applied during memory reactivation, later recall of that information is often stronger ([Javadi & Cheng, 2013](#)).

These and other reconsolidation studies illustrate that memory is not static, like a printed record, but dynamic. Each time we retrieve an old memory, we can modify it by integrating new information into it ([Dudai, 2004](#); [Sara, 2000](#); [Wang & Morris, 2010](#)). This allows existing memories to be continually updated, refined, and elaborated based on new experiences—but it also opens the door to potential disruption ([McKenzie & Eichenbaum, 2011](#); [Tronson & Taylor, 2007](#)). In fact, reconsolidation may be a key mechanism that underlies the creation of false memories. Each time a person remembers or re-imagines an event, that memory may be subject to modification, allowing minor distortions to creep in; over time, the distortions may become indistinguishable from the real facts of a case.

Metamemory

Suppose you were given a pop quiz, right now, on the material in this chapter so far. How well do you think you would do? **Metamemory** refers to knowledge or belief about one's own memory, and it applies to both episodic memory and semantic memory.

metamemory

Knowledge of, and ability to think about, our own memories, including both feeling of knowing and judgment of learning.

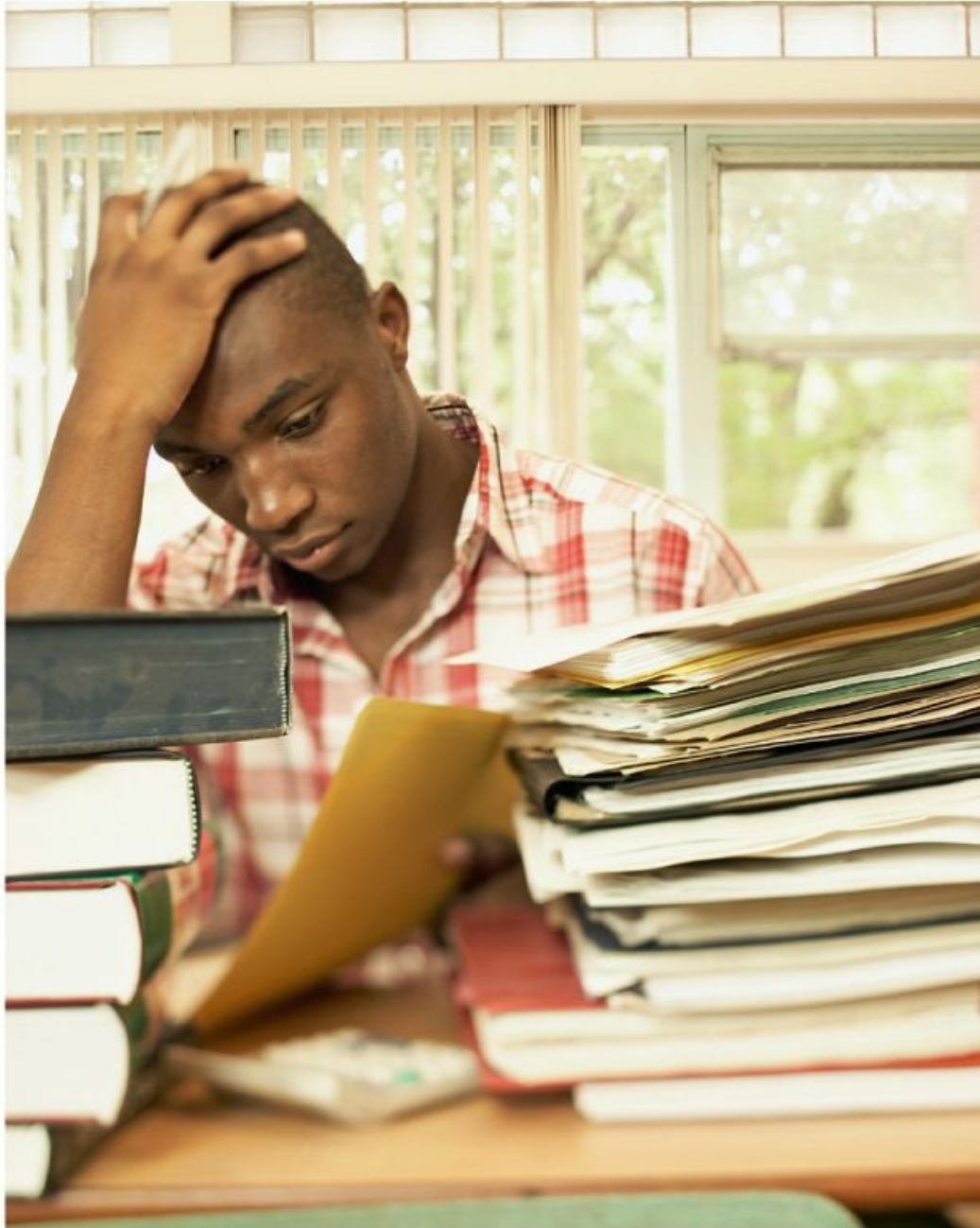
You have already read about one familiar example illustrating metamemory: the *tip-of-the-tongue* (TOT) phenomenon, in which we are challenged to retrieve a piece of information (such as the name of a film star or the answer to a test question) and feel sure it is something we know, even though we cannot retrieve it at the moment (“It’s on the tip of my tongue!”). Often, once we hear it, we “recognize” it—because, at some level, we did already know it. On the other hand, sometimes we are asked a question and we know that we *do not* know the answer. For instance, if someone asks you the atomic number of the chemical element boron, you may immediately know that you do not know the answer, no matter how long you might be given to try to recall the information.

A formal name for such phenomena is *feeling of knowing* (FOK), which refers to our ability to

predict whether or not we can retrieve a specific piece of information if asked. FOK judgments are not always accurate. Usually, FOK errors reflect overconfidence: a person may report feeling that he knows the answer to a question even when he does not (e.g., [Nelson, Gerler, & Narens, 1984](#)). One factor contributing to FOK errors is familiarity with the cue: the feeling that one “should” know the answer may lead to an illusion that one actually does know the answer. Thus, someone who recently completed a chemistry course may be more subject to an FOK error regarding the atomic number of boron than someone who never studied the topic and has no reason to believe he “should” know the answer.

A related metamemory phenomenon is *judgment of learning (JOL)*, which is a judgment during learning of whether the information has been successfully acquired. For example, a student studying for an exam has to decide when she can stop studying because the material to be tested is securely in her memory. Like FOK, JOL can be extremely inaccurate—again, usually in the direction of overconfidence. One factor is that people tend to assume that they will always remember information as well as they remember it right now. In one particularly striking study, participants studied lists of word pairs and were asked to predict how well they would remember the pairs if tested immediately, if tested 1 day later, or if tested 1 week later ([Koriat, Bjork, Sheffer, & Bar, 2004](#)). Most participants predicted that they would recall the information equally well in each case; in actuality, as could be predicted from [Figure 7.6A](#), the participants’ recall fell sharply as the delay between learning and testing increased.

One factor contributing to JOL errors is that people are relatively poor at judging the most effective ways to learn new information (for review, see [Bjork, Dunlosky, & Kornell, 2013](#)). For example, as you read earlier, self-testing typically strengthens memory much more than mere rereading does. But in one poll of college students, only 11% reported using self-tests while studying. Even among those students who did self-test, only 18% said that they did it because they learn more when they self-test than when they reread; the rest said that they used self-testing to assess how well they had learned the information ([Kornell & Bjork, 2009](#)). JOL errors can lead students to feel overconfident in their familiarity with course material, which is why students can sometimes be honestly surprised at poor performance on an exam when they feel they studied hard to prepare. (For more information on how to use this and other declarative memory research to your best advantage while studying, see [“Learning and Memory in Everyday Life”](#)).



Jupiterimages, Brand X Pictures/Getty Images

What kinds of metamemory errors might lead this student to stop studying before he has really mastered the information?

LEARNING AND MEMORY IN EVERYDAY LIFE

Optimize Your Study Habits

Most college students report that they study for exams primarily by rereading their textbook and notes ([Putnam, Sungkhasettee, & Roediger, 2016](#)). And indeed, rereading the material can be useful. Yet, as you now know, repetitious

passive exposure is not necessarily the best way to strengthen memory. Instead, here are some concrete ways to apply memory research findings to optimize your study habits. (For more on the science behind these strategies, see [Miyatsu, Nguyen, & McDaniel, 2018](#), and [Putnam et al., 2016](#).)

- If your textbook offers comprehension questions, try answering them *before* you read the chapter. Struggling to answer questions *before* you read makes you think about the topic. When you then read the chapter, you will be connecting the information you read to knowledge you have already retrieved about the topic. (Remember that learning is better if you can relate the new information to preexisting knowledge.)
- Also, *before* you read the chapter, take a few minutes to skim the pages and outline the contents. Noticing the general structure of the material you are about to read will help you organize the information as you are reading—and remember it later.
- Highlighting material in the textbook can be helpful, but it is best to do it *after* your first read-through, so you can better identify what is most important.
- Attend the lectures, even if the instructor mainly covers information that is also in the textbook. Exposing yourself to the material several times, in different formats, helps form a strong memory.
- In class, consider taking notes by hand, rather than typing into the computer. When typing, most students just try to quickly transcribe what they hear, and this shallow processing involves little thought about the meaning of the material being typed. In contrast, students writing by hand typically cannot keep up with every spoken word, so they must process the material more thoughtfully in order to choose what to write down. This deeper processing helps form strong memories that can be expressed flexibly at exam time.
- If your course offers practice quizzes, perhaps in an online resource, use them! Challenging your memory encourages deep thinking; and struggling to remember in itself can improve your memory. If no prepackaged quizzes are available, make up your own questions and try to answer them.
- While studying for exams, be alert for feeling-of-knowing misjudgments. Even if the material “feels” familiar, you may not know it as well as you think you do.
- Get a good night’s sleep the night before the exam. Sleep allows your brain to organize and consolidate materials from the day’s studying, and it also improves your attention and memory retrieval the next day. (You will read more about sleep in [Section 7.2](#).)

Interim Summary

- Episodic memory is personal memory for specific events that were experienced in a unique spatial and temporal context; it is information we “remember.”
- Semantic memory is memory for facts about the world and about ourselves. It does not necessarily include information about where or when the memory was originally acquired; it is information we “know.”
- Both episodic and semantic memories can be flexibly communicated in ways other than those in which they were originally acquired, and both are available to conscious recollection.
- Researchers still debate whether nonhuman animals can have true episodic memories; some believe this faculty belongs to humans alone, but others cite mounting evidence for “episodic-like” memory in nonhuman species.
- Several principles govern the encoding of new memories: (1) mere exposure to information

does not guarantee memory; (2) memory for new information is stronger if it can be related to existing knowledge; and (3) memory is also affected by how deeply the information is processed during encoding.

- Successful retrieval of existing memories is more likely (1) if encoding and retrieval conditions match (encoding specificity principle), (2) if processing is similar at encoding and testing (transfer-appropriate processing), and (3) if more cues are available to prompt recall.
- Struggling to retrieve material (such as during a test) often promotes longer-lasting memories than simply studying the material again.
- Memory can “fail” for many reasons, including simple forgetting, interference, source monitoring errors, and creation of false memories.
- Semantic and episodic memories appear to be subject to a time window called the consolidation period during which they are vulnerable and easily lost. Each time a memory is accessed, it may become vulnerable again and need to be reconsolidated.
- Metamemory refers to knowledge or belief about one’s own memory, and it includes feeling of knowing (illustrated by the tip-of-the-tongue phenomenon) and judgment of learning (such as when a student decides he has mastered the material well enough to stop studying). People are not always very accurate at evaluating or predicting their own memory.

7.2 Brain Substrates

Functions as complex as semantic and episodic memory depend on many interacting brain systems, and scientists are still a long way from fully understanding them all. Fortunately, new research is giving us a much clearer picture of how declarative memories are encoded, stored, and reactivated in the brain.

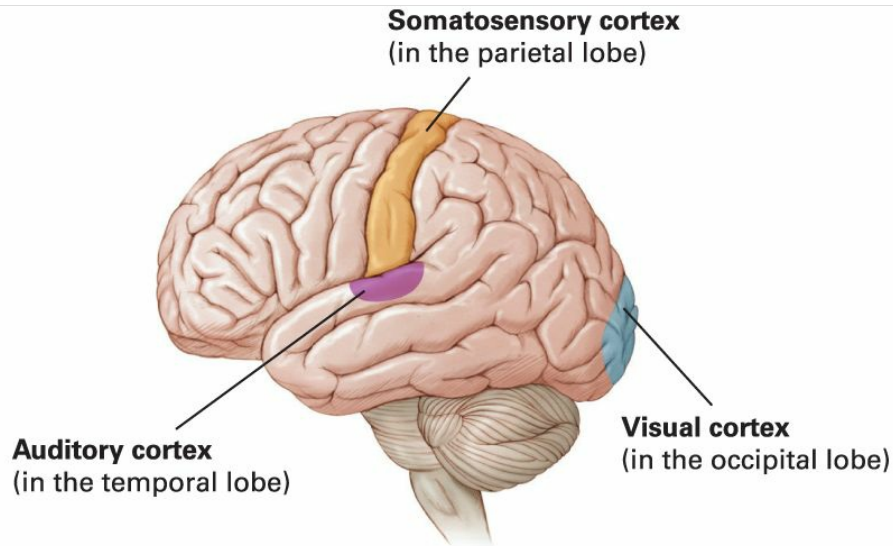
Cortical Networks for Semantic Memory

As summarized in the neuroanatomy discussion in [Chapter 2](#), the cerebral cortex can be divided into different regions, some of which preferentially process particular kinds of sensory information. These include the somatosensory cortex in the parietal lobe, the visual cortex in the occipital lobe, and the auditory cortex in the temporal lobe. Such sensory-specific parts of the cortex are collectively referred to by the general name [sensory cortex](#). Some research suggests that specific types of semantic knowledge are stored in the cortical areas devoted to processing the corresponding kind of information, meaning visual information would be stored in the occipital lobe, auditory information in the superior temporal lobe, and so on.

sensory cortex

Areas of the cerebral cortex involved in processing sensory information such as sight and sound.

Other cortical areas (e.g., many of the areas shown in pale pink in [Figure 7.10](#)) are called [association cortex](#), meaning they play a role in the brain's ability to form associations of information within and among modalities. For example, consider everything you know about the semantic concept of “dog.” Association cortex may help us link memories for the sound of dogs barking (which may be stored in auditory cortex) with visual images of dogs (which may be stored in visual cortex) and with the way their fur feels (somatosensory cortex) and smells (olfactory cortex), as well as with linguistic information about how to pronounce and recognize the word “dog.”



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

FIGURE 7.10 Semantic memory and the cerebral cortex Some areas of the cerebral cortex specialize in processing specific kinds of sensory information; these include areas in the parietal lobe (somatosensory cortex), the occipital lobe (visual cortex), and the superior temporal lobe (auditory cortex). Many of the remaining cortical areas are association areas that link information within and among modalities, forming a basic substrate for semantic information.

Description

The parts labeled are as follows. Somatosensory cortex (in the parietal lobe) lies like a band across the top of the parietal lobe. Auditory cortex (in the temporal lobe) on the superior temporal gyrus, below the somatosensory cortex. Visual cortex (in the occipital lobe) lies at the rear of the occipital lobe

association cortex

Areas of the cerebral cortex involved in associating information within and across sensory modalities.

Single Neurons Respond Selectively to Specific Stimuli or Categories

Some studies suggest that specific categories of semantic information are encoded by specific groups of cortical neurons. For example, in one study, Edmund Rolls and colleagues ([Thorpe, Rolls, & Maddison, 1983](#)) recorded neurons in the orbitofrontal cortex of rhesus monkeys. (This is a small area located at the base of the frontal lobes, described in [Chapter 5](#) and shown in [Figure 5.9](#).) Out of 494 neurons analyzed, Rolls and colleagues found 26 neurons that responded selectively to visual stimuli representing food. Even more striking, 4 of those neurons responded most strongly to oranges, 4 responded to peanuts, 2 responded to bananas, and 1 responded to raisins.

Similar specificity can be observed in humans. Sometimes patients who need brain surgery have intracranial electrodes implanted in preparation for the surgery, and some of these patients agree to undergo memory testing while the electrodes are in place. In certain studies, the patients are shown pictures, and the responses of individual neurons to each picture can be recorded. Some neurons respond to many pictures; others respond to none. Some respond to pictures of particular categories of objects, such as animals, faces, or houses ([Krieman, Koch, & Fried, 2000](#)). Some respond to sad faces but not happy, angry, or neutral faces ([Fried, MacDonald, & Wilson, 1997](#)).

In one striking study, a single neuron from one patient fired strongly in response to photos of actor Steve Carell but was nearly silent in response to photos of other celebrities, such as Whoopi Goldberg and Bill Clinton. Another patient had a neuron that responded to photos of Jennifer Aniston and Lisa Kudrow, both of whom were actresses in the TV series *Friends*; a different neuron in the same patient responded to pictures of the Eiffel Tower and the Tower of Pisa but not to other famous landmarks, such as the Golden Gate Bridge and the Sydney Opera House ([Quiroga, Reddy, Kreiman, Koch, & Fried, 2005](#)).

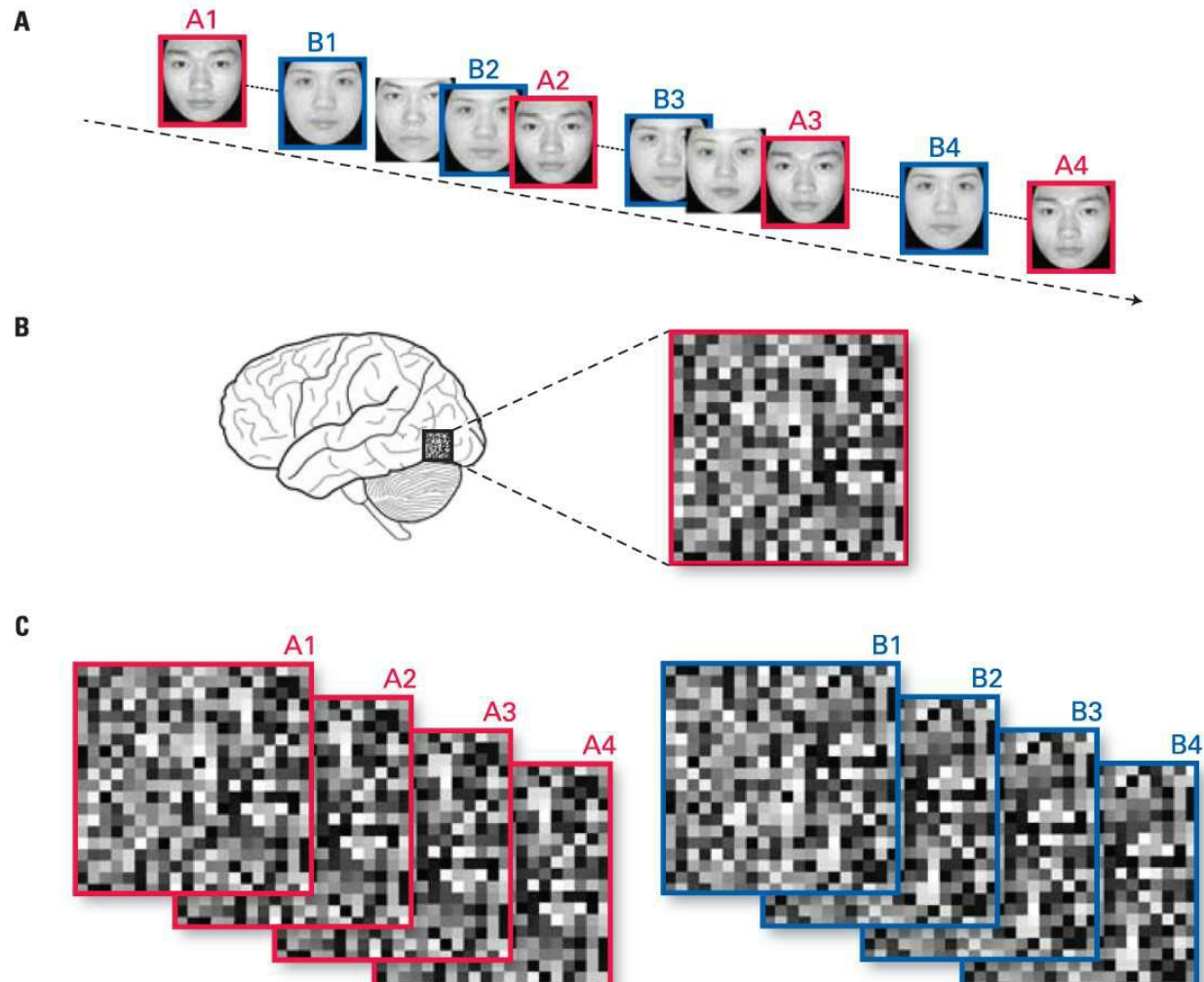
Remarkable as these results are, the fact that a neuron responded “only” to Steve Carell in the experiment does not mean that we have individual neurons specialized to detect images of individual people (one neuron for Steve Carell, one for Whoopi Goldberg, one for your grandmother, and so on). In fact, there might be many other people or objects—not explicitly tested in the experiment—that would also elicit a response from that neuron. Most researchers believe it is more likely that cortical neurons are associated in networks that respond to information representing simple, familiar categories (such as TV-sitcom actors, large pointy landmarks, or food items) and that each item within such a category is represented by a distinguishable pattern of responding across the category’s network of thousands (or even tens of thousands) of neurons. [Chapter 2 \(Figure 2.7\)](#) presents the idea of Hebbian learning, in which “neurons that fire together wire together,” forming networks that represent information as a pattern of activity across a number of neurons. These cortical neurons may be elements in such networks.

Neuronal Networks Encode Mental Representations

What do the networks that represent semantic information look like in healthy humans?

Recently, an approach called *representational similarity analysis*, or RSA ([Kriegeskorte, Mur, &](#)

[Bandettini, 2008](#)), has given us important clues. One version of RSA uses high-contrast fMRI to compare cortical responses to different events (for review, see [Xue, 2018](#)). In these studies, participants repeatedly view a series of stimuli, such as images of a few faces ([Figure 7.11A](#)). The first time each stimulus is viewed, it evokes a specific pattern of activity in several brain areas, typically including areas of sensory and association cortex. For example, if the participant is viewing images of faces, researchers might focus on the *left fusiform gyrus* (as shown in [Figure 7.11B](#)), which is an area of visual association cortex that is important for facial recognition. The fMRI data from numerous tiny subareas in the selected brain area are then analyzed and used to produce a pattern, as schematized in [Figure 7.11B](#). [Figure 7.11C](#) shows that when the participant views the same face again, a similar activity pattern is evoked, whereas other faces evoke different patterns ([Xue et al., 2010](#)). Another participant's data would generate a different pattern still—one that also is similar for that person's multiple views of the same face but different from the patterns evoked by other faces.



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FIGURE 7.11 Memory representation in the cortex (A) To study how information is represented across large numbers of neurons in the cortex, researchers conduct high-resolution fMRI scanning while participants repeatedly view a series of stimuli (such as images of faces "A" and "B"). Here, face A and face B are each viewed several times. (B) The fMRI readouts provide a "pattern" of activity in a target brain region. One hypothetical pattern ("map") is presented here, showing the activity in a region of visual association cortex in response to the first viewing of face A. (C) The activity patterns from each viewing of face A (maps A1 through A4) are highly correlated with each other but much less correlated with patterns from the viewing of face B (maps B1 through B4). The more highly correlated the patterns generated on each viewing of a stimulus, the better the memory for that stimulus ([Xue et al., 2010](#)).

Description

Illustration A shows ten photographs of a two faces are arranged horizontally, of which the photos are labeled as follows: A 1, B 1, (not labeled), A 2, B 2, B 3, (not labeled), A 3, B 4, and A 4. Illustration B shows an outline of

human brain, in which a magnified image from the occipital lobe shows the brain activity pattern. Illustration C shows four brain activity patterns for A 1, A 2, A 3, and A 4, and four brain activity patterns for B 1, B 2, B 3, and B 4.

When the participant is asked to recall a face from memory, a pattern is evoked that is very similar to the one produced when the face was actually being viewed ([Larocque et al., 2013](#)). Further, the more consistent a stimulus's cortical representation across multiple viewings, the better the subsequent memory for that stimulus will be ([Xue et al., 2010](#)). In short, activation patterns like those in [Figure 7.11](#) give us a glimpse into how the participant's brain is representing—or coding—the visual appearance of each face. Similar results have been seen in other brain areas when participants are viewing other types of stimuli, such as words or inanimate objects.

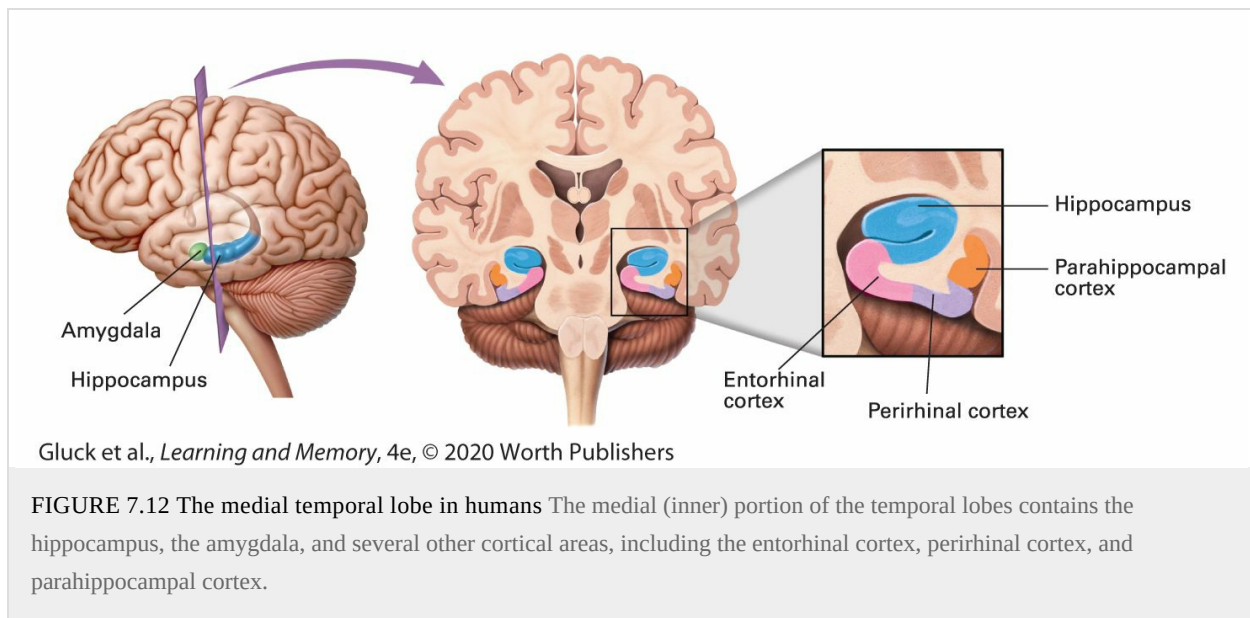
Of course, a rich semantic memory for a person—whether someone you know or a celebrity such as Steve Carell—contains more than just visual information about the face. It also contains memory about the person's name, the sound of his or her voice, the person's personality, names of movies starred in, and so on—all of which would be represented as specific activation patterns in other areas of association cortex. These activity patterns are presumably linked together across multiple cortical areas, such that when we see the person's face, thereby triggering the corresponding activity pattern in our facial fusiform area, all the other associated aspects of a rich semantic memory, stored in multiple cortical areas, are similarly reactivated and available for recall.

Back in the 1920s, Karl Lashley conducted a frustrating search for the *engram*, or memory trace, the hypothesized physical change representing a memory (see [Chapter 2](#)). We cannot yet say we have found it, since each subarea visualized in a high-resolution fMRI image still represents the combined activity of large numbers of neurons—but this work is bringing us much closer to visualizing it.

The Medial Temporal Lobes and Memory Storage

The medial temporal lobes in humans contain the [hippocampus](#), the amygdala, and nearby cortical areas, including the entorhinal cortex, the perirhinal cortex, and the parahippocampal

cortex ([Figure 7.12](#)). [Chapter 3](#) describes the role of the hippocampus in spatial learning, and [Chapter 4](#) describes the role of the hippocampus in certain forms of classical conditioning. But, in fact, the most widely studied role of the human hippocampus is in the storage of new declarative memories, and the most obvious impairment in humans with damage to the medial temporal lobe is the loss of this function.



Description

The diagram on the left has parts labeled as follows. Amygdala and Hippocampus, at the tip of the brain. The diagram in the middle shows an enlarged coronal cross-section view of Entorhinal cortex. The diagram on the right shows the enlarged view of the hippocampus, at the inner part of a spiral attached to the entorhinal cortex, which is connected to the perirhinal cortex and parahippocampal cortex.

hippocampus

A brain structure located in the medial temporal lobe that is important in the formation of new memories.

At the start of this chapter, you read about H.M., the man who became amnesic after bilateral surgical removal of his medial temporal lobes ([Scoville & Milner, 1957](#)). Earlier, other neurologists in the late 1800s and early 1900s had noted the role of the medial temporal lobes in memory. But H.M.'s case provided especially strong evidence of their function—partly because of the severity of his impairment and partly because of H.M.'s decades-long willingness to participate in all kinds of memory tests that carefully documented exactly what he could and could not learn and remember.

Obviously, after H.M., no more surgeries were performed to remove the medial temporal lobes bilaterally in humans (although unilateral surgeries, which do not cause severe amnesia, are still sometimes performed for patients with intractable epilepsy). Unfortunately, bilateral medial temporal lobe damage does occur in humans as a result of various other kinds of injury and disease, and these patients often show a pattern of memory impairment very similar to that exhibited by H.M.

The Hippocampus and Encoding of New Episodic Memories in Humans

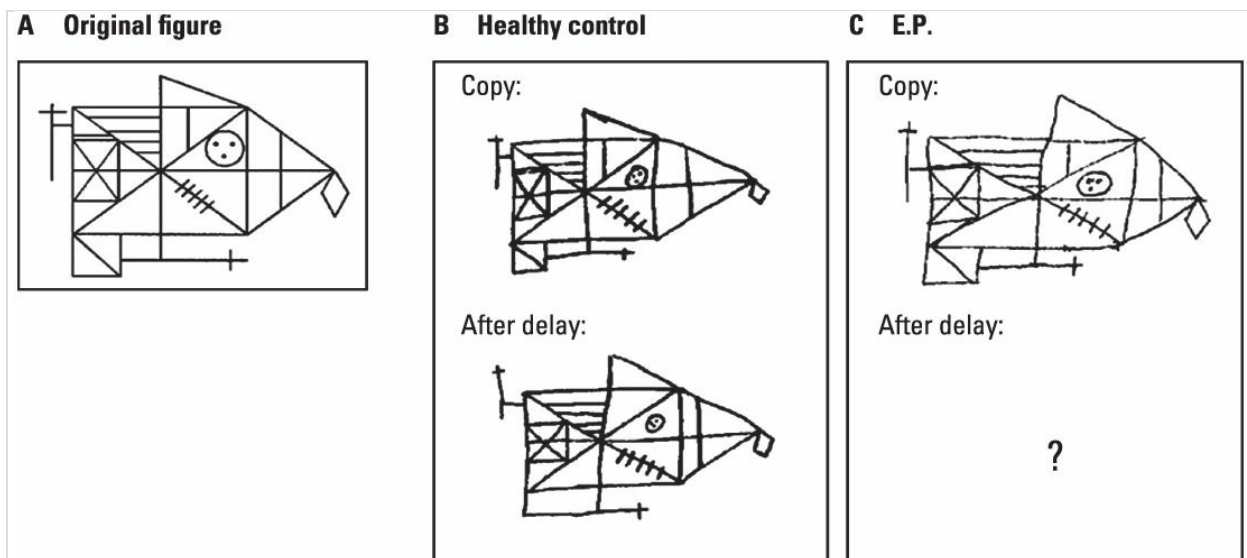
The most profound and noticeable memory impairment in patients such as H.M. is [anterograde amnesia](#), a severe impairment in the ability to form new declarative memories. Patients with bilateral hippocampal damage are particularly impaired at forming new episodic memories of personal events, learned after a single exposure and tagged in time and space. For example, Larry Squire and colleagues studied one patient, known by his initials E.P., who suffered bilateral medial temporal damage as a result of viral encephalitis ([Stefanacci, Buffalo, Schmolck, & Squire, 2000](#)). When questioned about autobiographical events, E.P. could report details of his childhood, his wedding, and his travels as a young man. But he had little or no ability to form new episodic memories: he could not recall what he had done yesterday or almost anything that had happened to him since the onset of his amnesia several years previously. Within a 1-hour session, he might repeat the same anecdotes over and over as many as 10 times, unaware that he had told the stories before ([Stefanacci et al., 2000](#)).

anterograde amnesia

A severe loss of the ability to form new declarative memories.

Like H.M., E.P. participated in a large number of memory tests. One standard test of declarative memory uses the complex figure shown in [Figure 7.13A](#). Participants are first asked to copy the figure and then to draw it from memory 10 to 15 minutes later. Healthy adults can usually copy the figure quite accurately and manage to reproduce the figure from memory reasonably well after the delay ([Figure 7.13B](#)). E.P. could copy the drawing without difficulty, but after the delay, he had no memory of the picture at all ([Figure 7.13C](#); [Insausti, Annese, Amaral, & Squire, 2013](#)). His memory for verbal information was similarly poor: immediately after listening to a short story, he could recall only about 10% of the words or concepts; 15 minutes later, he could remember nothing about it. Studies from patients such as H.M. and E.P. show that declarative

memory, and particularly episodic memory, is dependent on the hippocampus and nearby structures in the medial temporal lobe.



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

FIGURE 7.13 Anterograde amnesia in patient E.P. Given a complex figure (A), healthy controls can usually copy it accurately (B, top); 15 minutes later, they can still produce a reasonably accurate drawing from memory (B, bottom). Patient E.P. could copy the drawing about as well as healthy controls (C, top). However, when asked to draw the figure from memory 15 minutes later, E.P. did not recall making the copy and declined to try to draw it from memory.

From [Insausti et al., Human amnesia and the medial temporal lobe illuminated by neuropsychological and neurohistological findings for patient E.P. Proc Natl Acad Sci U S A. 2013 May 21;110\(21\):E1953-62](#)

Description

The illustration labeled Original figure incorporates lines, triangles, a face, and multiple sections. The illustration labeled Healthy control has two diagrams, Copy and After delay. The illustration labeled E.P has two diagrams, Copy and After delay. The figure under Copy has a complicated figure, and the area under After delay shows a question mark.

Our understanding of the human hippocampus is not based only on patients with amnesia. Functional neuroimaging studies allow us to see the hippocampus in action in brains with normal memory function. For example, in the *subsequent memory* paradigm, developed by Anthony Wagner and his colleagues (e.g., [Wagner et al., 1998](#)), participants first view a list of words while in an fMRI scanner. During this phase, the participants complete some task involving the words, such as judging whether the words represent animate or inanimate objects. Later, the researchers administer a surprise recognition test, showing the participants a new list of words

and asking them to identify which words were on the previous list. As you would expect, people correctly recognize some of the words but not all of them. The important finding is that the hippocampus is generally more active during initial study of words that are subsequently remembered than of words that are subsequently forgotten ([Otten et al., 2001](#)).

There is a similar correspondence when participants view pictures that will later be remembered versus those that will later be forgotten ([Brewer, Zhao, Desmond, Glover, & Gabrieli, 1998](#)). In effect, researchers examining the fMRI of a participant's brain during learning can predict with reasonable accuracy which pieces of information will be recalled or forgotten later ([Wagner et al., 1998](#)).

So what exactly is the hippocampus doing when we successfully (or unsuccessfully) store new memories? Just as with cortex, researchers can use high-resolution fMRI and representational specificity analysis to examine patterns of neuronal activation in the hippocampus during learning. In one study, Lila Davachi and colleagues showed participants pairs of photographed objects, such as rubber duck and rose, wrench and basketball, chair and rose, and so on ([Tompary, Duncan, & Davachi, 2016](#)). Later, the participants were shown single objects and were asked to recall the other member of the pair (e.g., rubber duck and _____). Each pair was shown only once, although some objects appeared in more than one pair.

Unsurprisingly, when being tested, participants correctly recalled some of the paired objects but had forgotten others. More importantly, the researchers compared activation patterns in the hippocampus for each pair of objects during encoding and during recall. For object pairs that were successfully recalled, the activation patterns in the hippocampus were quite similar at encoding and recall; but for forgotten pairings, the activation patterns in the hippocampus were much less similar at encoding and recall ([Figure 7.14](#)). The nearby perirhinal cortex showed a similar effect; other brain areas that are not thought to play a role in episodic memory (such as parts of the basal ganglia) showed no such effect.

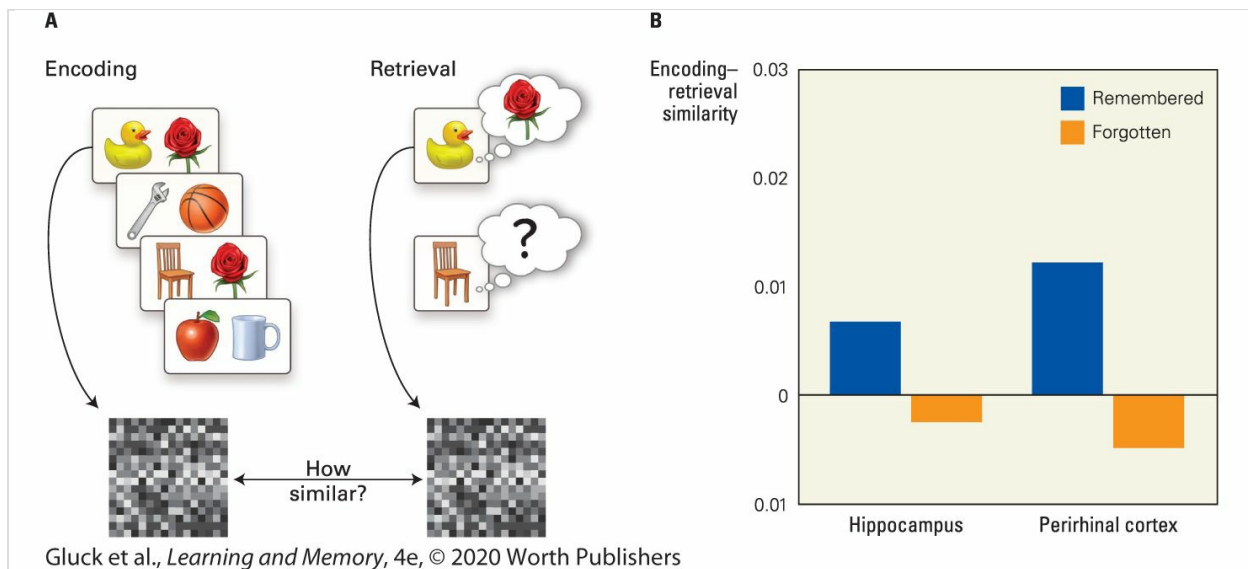


FIGURE 7.14 Memory representation in the hippocampus (A) During the encoding phase, participants view pairs of objects; for each pair, high-resolution fMRI shows activation patterns in the hippocampus. A hypothetical pattern for the rubber duck–rose pair is shown here. Later, during retrieval, participants view one object from each pair and are asked to recall the associated object; again, high-resolution fMRI is used to collect activation patterns on each trial. Activation patterns for each object pair can then be compared for similarity at encoding versus retrieval. (B) In the hippocampus and also in the nearby perirhinal cortex, encoding and retrieval activation patterns are more similar for correctly remembered pairs than for forgotten pairs (Tompary et al., 2016). This suggests that successful memory retrieval occurs when the hippocampus rapidly encodes information that can be faithfully retrieved later.

Information from Tompary et al., 2016.

Description

The illustration labeled A shows Encoding and Retrieval. It shows a pair of objects during the encoding phase form a hypothetical pattern of the rubber duck and rose, screw tightener and baseball, chair and rose, and apple and a mug. It shows a pair of objects during the retrieval phase forms a hypothetical pattern if rubber duck and rose, and chair and a question mark. Both the phases point to an fMRI resolution pattern. The illustration labeled B shows a graph. The X-axis shows Hippocampus and Perirhinal cortex, and Y-axis shows Encoding–retrieval similarity. The data in the graph are as follows. Hippocampus: 0.85. Hippocampus: -0.99. Remembered: 0.02. Perirhinal cortex: -0.85.

These and related results strongly suggest that the hippocampus plays a critical role in the rapid encoding of memory for information that is encountered only once—and that the success or failure of later memory retrieval at least partially reflects how reliably these patterns can be reactivated later, as well as how similar or different they are from the patterns evoked by other, competing memories.

The Hippocampus and Encoding of Episodic-Like

Memories in Nonhuman Animals

Much of what we have learned about the hippocampus in humans applies equally to nonhumans. Just like H.M., animals with broad lesions of the hippocampal region have difficulty learning new information ([Mishkin, 1978](#); [Squire, 1992](#)). They are especially impaired at episodic-like learning that involves memory of unique events set in a particular context ([Gaffan & Hornak, 1997](#); [Gaffan & Parker, 1996](#)). For example, the radial maze shown in [Figure 7.2](#) can be used for a task in which researchers place a piece of food at the end of every arm and then place a hungry rat in the center of the maze. The rat has a few minutes in which to obtain all the food it can. Notice that once a rat has entered a given arm and eaten the food there, subsequent entries into the same arm are a waste of time. Therefore, the rat can solve this task most efficiently by visiting each arm exactly once. This requires that the rat remember where it has already been. But if the same task is repeated on several successive days, the rat will have to remember which arms it has already visited so far today, distinct from the memories of prior days on which the rat entered those arms and found food there. Proactive interference affects rats in a radial arm maze just as it affects humans in a parking lot!

The only way the rat can solve this dilemma is by remembering the spatial and temporal contexts of its visits to the various arms—a form of episodic-like learning. After several days of training, healthy rats learn to navigate the radial arm maze very efficiently: they collect all eight rewards, and in the process, they make very few erroneous reentries into previously visited arms. In contrast, rats with hippocampal lesions make many more errors. These rats repeatedly reenter previously visited arms, indicating semantic memory that there is food to be found, but without episodic-like memory for which specific arms they have already visited on a particular day ([Cassel et al., 1998](#); [Jarrard, Okaichi, Steward, & Goldschmidt, 1984](#); [Olton, 1983](#)).

In birds, hippocampal-region damage also disrupts episodic-like learning. Remember the scrub jays, who bury food in caches and then return to the caches later? When the hippocampal region of a scrub jay is lesioned, the bird loses the ability to locate its caches ([Capaldi, Robinson, & Fahrback, 1999](#)). It continues to store new food but quickly forgets where it has put it; later, the bird searches almost at random—much like a lesioned rat running around a radial maze.

The Medial Temporal Lobes and Encoding of New Semantic Memories

While most researchers agree that the hippocampus is critical for the acquisition of new episodic memories, which are tagged with a time and place, there is less certainty about the acquisition of new semantic memories. Many studies suggest that acquisition of new semantic memories depends less on the hippocampus than on other medial temporal lobe structures ([Diana, Yonelinas, & Ranganath, 2007](#); [Eichenbaum, Yonelinas, & Ranganath, 2007](#)), such as the perirhinal cortex or parahippocampal cortex (see [Figure 7.12](#)). Thus, although patient H.M. had profound anterograde amnesia, he could learn new semantic information after laborious study. For instance, when asked about John F. Kennedy and Mikhail Gorbachev, who each became famous after the onset of H.M.'s amnesia, H.M. could correctly respond that Kennedy had been president and that someone had shot him and that Gorbachev had been head of the Russian parliament ([O'Kane, Kensinger, & Corkin, 2004](#)).

In contrast, patient E.P., whose damage extended further into the medial temporal lobe than H.M.'s, was largely unable to acquire new semantic information. A year after the onset of E.P.'s amnesia, he and his family moved to a new home in California; but after 7 years of living there, E.P. could not draw a floor plan of the new home, describe how to get to the grocery store, or even point in the direction of the Pacific Ocean, which was less than 2 miles from his home ([Stefanacci et al., 2000](#)). In general, patients with extensive damage to the temporal lobes perform poorly on tests of semantic memory, whereas patients with damage limited to the hippocampus tend to have much more modest semantic memory impairments ([Insausti et al., 2013](#)).

Other species, such as birds, rats, and monkeys, also have brain regions analogous to the human parahippocampal cortex and perirhinal cortex; for simplicity, we will call these “parahippocampal areas,” although the regions have different names in some species. In a wide range of species, semantic memory appears to depend more on these parahippocampal areas than on the hippocampus itself (see [Eichenbaum et al., 2007](#), for review). Remember that whereas episodic memory is information you “remember,” semantic memory is information you “know” (or can recognize). Rats with hippocampal lesions can still recognize novel objects and spatial locations, indicating that they “know” what they have (and have not) seen before—but these abilities are devastated after lesions to parahippocampal areas. On the other hand, if the tasks require that the animals “remember” specific combinations of object and spatial location or temporal order (episodic-like memory for what happened where and when), the hippocampus is

required.

Determining What Gets Stored

So far in [Section 7.2](#), we have discussed long-term storage of semantic memories in the cortex, as well as the roles of various structures in the medial temporal lobes. Specifically, the hippocampus is important for encoding new episodic memories, while other medial temporal lobe structures (such as perirhinal cortex and parahippocampal cortex) are perhaps more important for learning new semantic information. But what determines which information should be encoded and where it should be stored? Two brain regions that play roles are the frontal cortex and a group of subcortical structures known collectively as the *basal forebrain* ([Figure 7.15](#)).

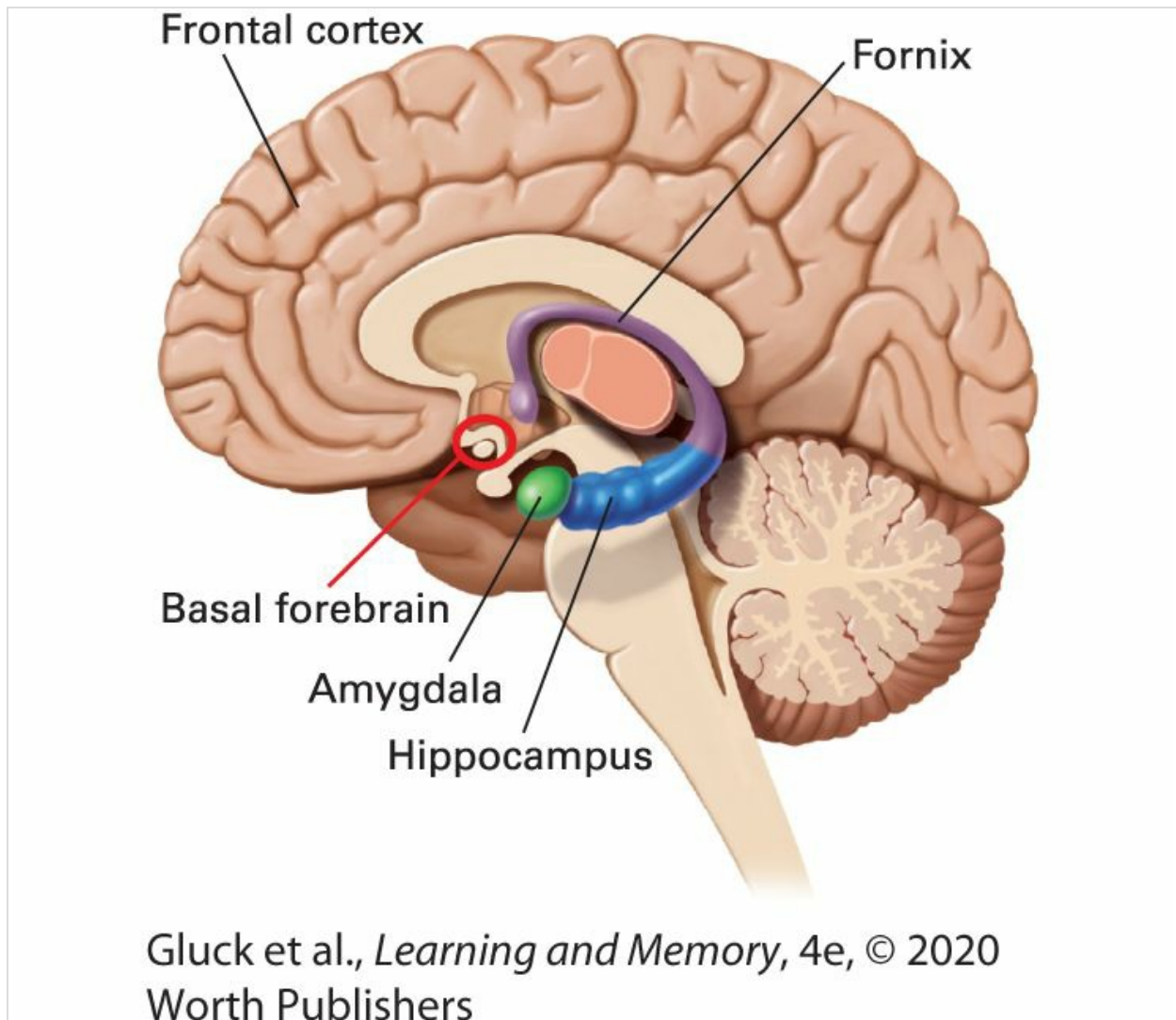


FIGURE 7.15 The frontal cortex and basal forebrain The frontal cortex projects to the hippocampus and may help determine what new information gets encoded as episodic and semantic memories and what gets forgotten; it may also play a part in binding contextual information to event memory. The basal forebrain is a group of subcortical structures that connect with the hippocampus via a fiber bundle called the fornix. Damage to the basal forebrain can cause anterograde amnesia that resembles the effects of direct hippocampal damage.

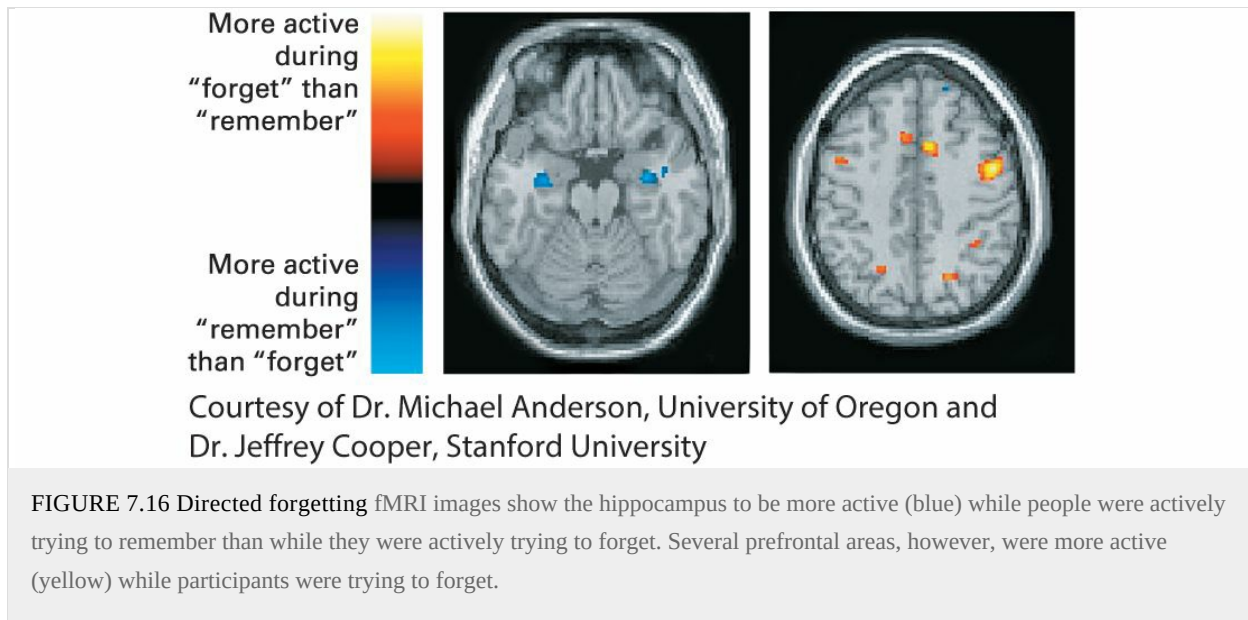
The Frontal Cortex

The [frontal cortex](#), those regions of cortex that lie within the frontal lobes, may help determine what information we store—and what information we forget. Some of the most compelling evidence for this idea comes from studies of directed forgetting. An example of the directed forgetting effect was shown in [Figure 7.6B](#), where participants' memory was worse for items they had been instructed to forget than for items they had been instructed to remember (or for items they had not seen since the original study phase).

frontal cortex

The regions of cortex that lie within the frontal lobes and that may play a role in determining which memories are stored and in producing metamemory for that information.

What could underlie this effect? Michael Anderson and colleagues collected fMRI data comparing brain activity while participants were actively trying to remember or forget words ([Anderson et al., 2004](#)). Just as you would expect, the hippocampus was more active while participants were actively trying to remember than when they were trying to forget. But several areas in the prefrontal cortex were more active while the subjects were intentionally trying to *forget* than when they were trying to remember ([Figure 7.16](#)). One possible explanation is that certain areas of prefrontal cortex inhibit the hippocampus, so their heightened activity during intentional forgetting suppresses hippocampal memory encoding. And, indeed, the greater the prefrontal activation in a participant, the more likely that participant was to forget on the final test.



Description

A color-coded scale on the left shows a wide range of colors and the scale marked from top to bottom reads, More active during forget than remember and More active during remember than forget. The photo on the right shows an fMRI image of the human brain, with the frontal cortex and basal forebrain highlighted.

These and similar experiments strongly suggest that the frontal lobes help determine what new information gets encoded as episodic and semantic memories and what gets forgotten. The frontal lobes may also help us bind contextual information to event memory, allowing us to form episodic memories that incorporate rich semantic information about where and when the events took place ([Schacter & Curran, 1995](#)).

In keeping with these ideas, you might expect that people with frontal-lobe damage would be especially prone to source monitoring errors—that is, unable to remember where and when an event occurred—and, indeed, this seems to be the case. Patients with frontal-lobe damage may be able to remember a story but not whether it occurred in their own past or on television or in their imagination ([Kapur & Coughlan, 1980](#)). Such source monitoring errors are generally not as devastating as the all-out memory failure seen in amnesic patients, but they can still be seriously problematic if the person cannot reliably tell the difference between imagined and real-life experiences.

The frontal cortex also plays an important role in metamemory, helping us keep track of what we

know and do not know. People with frontal-lobe lesions do poorly on tests of metamemory ([Pannu & Kaszniak, 2005](#)), and fMRI studies show that the right medial frontal cortex is active in healthy young adults who experience tip-of-the-tongue events in which they try and fail to retrieve information that they think they should remember ([Maril, Wagner, & Schacter, 2001](#)).

In sum, whereas the hippocampus and medial temporal lobes are key players in the encoding of new episodic and semantic information, the frontal cortex appears to be involved at a higher level, helping to determine what gets stored and what gets forgotten—and then helping us keep track of it all through metamemory.

The Basal Forebrain

Another brain area that helps govern information storage is the **basal forebrain**, a small collection of subcortical structures that lie—as the name suggests—at the base of the forebrain ([Figure 7.15](#)). The basal forebrain receives blood and oxygen from a small artery called the *anterior communicating artery* (ACoA). The ACoA is a common site of *aneurysm*, a type of stroke in which an artery wall balloons out under pressure and may even rupture. ACoA aneurysm rupture can cause damage to the basal forebrain, and survivors often have anterograde amnesia very similar to that caused by medial temporal lobe damage ([DeLuca & Diamond, 1995](#)).

basal forebrain

A collection of structures that lie at the base of the forebrain and are important in the production of acetylcholine that is distributed throughout the brain.

Why should basal forebrain damage cause amnesia? As described in [Chapter 2](#), neurotransmitters such as GABA and neuromodulators such as acetylcholine are critical for proper brain function. The *medial septum* is a group of cells in the basal forebrain, some of which produce the neurotransmitters acetylcholine and GABA. Axons from the neurons of the medial septum and other subcortical structures project to the hippocampus via an arch-like fiber bundle called (you guessed it) the **fornix**. These projections affect the activity and synaptic plasticity of hippocampal neurons and may help determine whether and when the hippocampus will process and store information ([Buzsaki & Gage, 1989](#); [Damasio, Graff-Radford, Eslinger, Damasio, & Kassell, 1985](#); [Hasselmo, 1999](#); [Myers, Ermita, Hasselmo, & Gluck, 1998](#)). This could explain why basal forebrain damage leads to amnesia: although the hippocampus is

undamaged, it cannot perform effectively without neuromodulation from the basal forebrain telling it when to store new information. By analogy, your computer may be in perfect working order, but if you disconnect your keyboard, it will be difficult to store any new files.

fornix

A fiber bundle that connects subcortical structures, including the basal forebrain, to the hippocampus.

One fascinating feature of basal forebrain amnesia is that when questioned about past events, patients may respond with highly detailed but false memories, a phenomenon called **confabulation**. For instance, asked what he did yesterday, a patient may say that he went into the office for a few hours, met an old friend for lunch, and then did some grocery shopping on the way home; the story may sound perfectly convincing except for the fact that the patient has been in the hospital for the past 2 weeks! Individuals who confabulate are not lying. Rather, they seem to believe the stories and are often sincerely confused when confronted with proof to the contrary.

confabulation

A behavior in which individuals, when asked to remember past events, respond with highly detailed but false memories; associated with some forms of *amnesia*.

Confabulation typically appears in patients in whom the damage from the aneurysm rupture extends beyond the basal forebrain and into the frontal cortex. And consistent with what we know about the role of the frontal cortex in memory, some examples of confabulation seem to be a kind of source monitoring error. In the above instance, the patient cannot remember what he did yesterday because of the amnesia produced by his basal forebrain damage. But he can retrieve a plausible answer from old (possibly semantic) memory. His frontal cortex is damaged, impairing metamemory and leaving him unable to determine whether the retrieved memory is old or new ([DeLuca, 2000](#)).

In many patients, confabulation is most severe immediately after the aneurysm but tends to decrease as time passes, possibly due to some recovery of function in prefrontal cortex. The amnesia, however, is generally permanent.

Many questions remain unanswered about the role of the basal forebrain and other subcortical structures contributing to memory storage. But the fact that anterograde amnesia can result from

so many different types of brain damage provides compelling evidence that memory is a function of the whole brain. Many structures—including the hippocampus, cortex, and basal forebrain—must all be working well and working together for episodic and semantic memory to function properly. Lashley would feel at least partially vindicated.

Long-Term Storage and Retrieval

An important feature of semantic and episodic memories is that they can last for years—or even a lifetime. You have already read about consolidation periods, during which new memories are highly vulnerable to change, and also about reconsolidation, the process whereby reactivation of an old memory renders it once again vulnerable to modification. How does this work in the brain?

Retrograde Amnesia

In the late 1800s, French philosopher Theodore Ribot noticed that individuals with head injury often developed **retrograde amnesia**, loss of memories for events that occurred before the injury ([Ribot, 1882](#)). This is in contrast to anterograde amnesia, in which patients lose the ability to form memories for events that occur after the injury. Typically, retrograde amnesia follows a pattern called the **Ribot gradient**, in which retrograde memory loss is worse for events that occurred shortly before the injury than for events that occurred in the distant past. The Ribot gradient also describes the effects of electroconvulsive shock, which preferentially disrupts recently formed memories (see [Figure 7.9](#)). As another example, a man who hit his head during a car accident might lose all memories of the accident itself and might also have some disruption of memories from the minutes or hours before the accident, but he would have relatively little disruption of memories for events that occurred months or years earlier.

retrograde amnesia

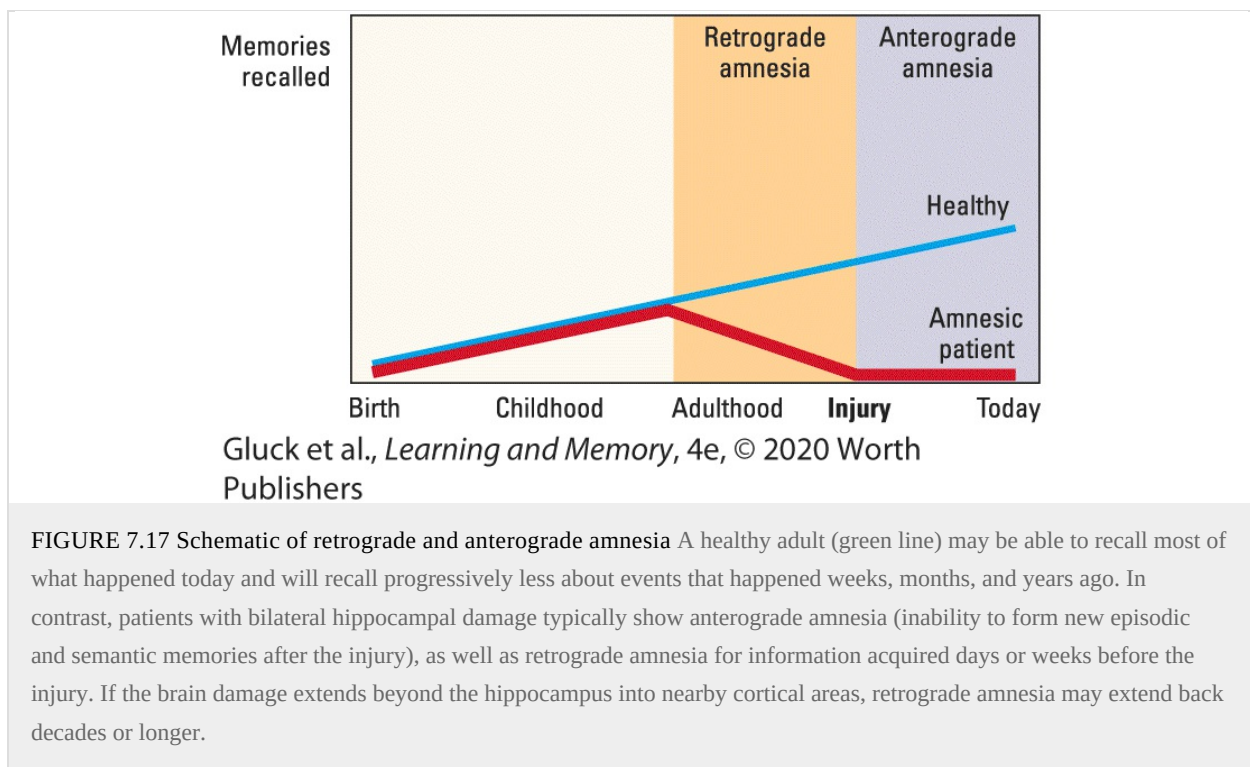
Loss of memories for events dating from before a brain injury or disruption; the memory loss is generally time graded, with more recent memories devastated and older ones being spared.

Ribot gradient

A pattern of retrograde memory loss in which recently acquired memories are more prone to disruption than older memories.

Patients with bilateral medial temporal lobe damage generally show some retrograde amnesia along with their anterograde amnesia ([Figure 7.17](#)). These patients do not forget their own identity—indeed, H.M. could remember his name and his childhood—but they often lose

memories for events that happened days or months before the brain damage, and this retrograde amnesia can affect information acquired decades earlier ([Manns, Hopkins, & Squire, 2003](#)). For example, as you read earlier, patient E.P. suffered bilateral medial temporal lobe damage that left him with dense anterograde amnesia, meaning that he could no longer store new declarative memories. In addition, E.P. displayed retrograde amnesia. His memory for childhood events was excellent—as good as that of healthy controls of the same age. But when asked about adulthood events that had occurred decades before his encephalitis, E.P. remembered significantly less than the controls ([Reed & Squire, 1998](#); [Stefanacci et al., 2000](#)).



Description

The Y-axis shows Memories recalled and X-axis is divided vertically into five sections as follows: Birth, Childhood, Adulthood, Injury, and Today. from left to right. The area shaded in the Adulthood section is labeled Retrograde amnesia. The area shaded in the portion between Injury and Today is labeled Anterograde amnesia. A straight line graph labeled Healthy slopes upward steadily. A line labeled Amnesic patient shows a slope moving upward, passing the sections Birth and Childhood. It suddenly declines to a lower point on the section Injury and angles down and stay at zero throughout the Anterograde Amnesia section.

E.P.'s loss of at least some memories of events that occurred many years in the past suggests that the consolidation period may last decades. If so, just how long is the consolidation period in

humans? How long before a new memory becomes independent of the medial temporal lobes and is “safely” stored in sensory and association cortex?

TEST YOUR KNOWLEDGE

Going Retro

The prefix *retro-* appears in two key terms in this chapter. First, you read about *retroactive interference*, when new information interferes with old memories. Later, you read about *retrograde amnesia*, when an event such as brain injury disrupts old memories. In each case, *retro-* means reaching back in time (think of “retro” fashions or “retroactive pay”). In contrast, *proactive interference* and *anterograde amnesia* both involve reaching forward in time: proactive interference occurs when old information reaches forward in time to disrupt future learning, and anterograde amnesia occurs when an event such as brain injury reaches forward in time to disrupt future formation of new memories. Can you correctly identify retroactive versus proactive interference and retrograde versus anterograde amnesia in the examples below? ([Answers appear at the end of the chapter.](#))

1. Scott took two semesters of Latin in college. He then spent a summer abroad in Spain and learned some Spanish while he was there. The next semester, he enrolled in another Latin course, but on his first test he mistakenly used some Spanish words instead.
2. Marina recently broke up with her longtime boyfriend Carl; last month, she started dating Lance. One night while she was tired and distracted, she called him Carl by mistake.
3. Ten years ago, John was involved in a motorcycle accident that caused damage to his medial temporal lobes, including his hippocampus. He spent 2 weeks in the hospital recovering before being released to the care of his wife at home. Now, John has no memory of the time he spent in the hospital.
4. John also has no memory of events that occurred in the months before his accident, although he remembers his childhood in Minnesota.

Standard Consolidation Theory

An early and influential idea, sometimes called [standard consolidation theory](#), holds that the hippocampus and related medial temporal lobe structures are required for the initial storage and retrieval of an episodic memory but that their contribution diminishes over time until the cortex is capable of retrieving the memory without hippocampal help ([Dudai, 2004](#); [McGaugh, 2000](#); [Squire & Alvarez, 1995](#)). This view conceptualizes an episodic memory as consisting of many components (sight, sound, texture, context, etc.) that are stored in different areas of the cortex ([Figure 7.18A](#)). Initially, all of these components are linked together via the hippocampus into a unified episodic memory ([Figure 7.18B](#)). Over time, through the process of consolidation, the components can form direct connections with each other—much like the Hebbian networks depicted in [Chapter 2](#)—and no longer need hippocampal mediation ([Figure 7.18C](#)). If the hippocampus is damaged, older memories would be more likely to have such connections, and

thus would be more likely to survive, than new memories. This explains the temporal gradient of retrograde amnesia observed in many amnesic patients with medial temporal lobe damage, such as H.M. and E.P.

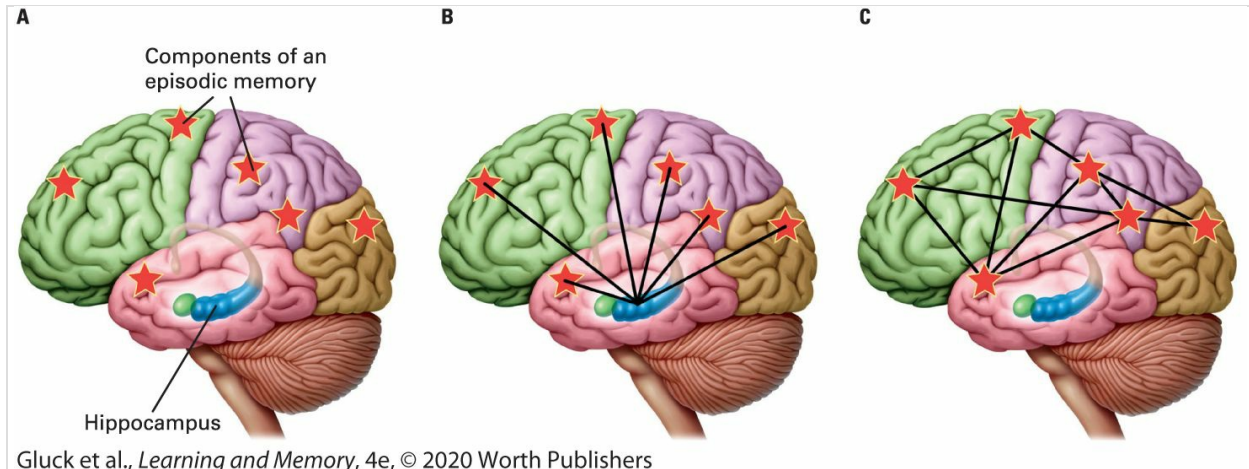


FIGURE 7.18 Theories of memory consolidation (A) An episodic memory consists of many components, such as sight, sound, texture, and other features, stored in sensory and association cortex. (B) Initially, the hippocampal region helps link these components into a single episodic memory. (C) Standard consolidation theory states that, over time, the components become linked to each other directly, and hippocampal involvement is no longer required. In contrast, multiple trace theory holds that, over many repeated episodes, common elements can be abstracted out to form a semantic memory, as depicted in part C, but that all episodic memories remain at least partially dependent on the hippocampus, as illustrated in part B.

Description

The illustration labeled A shows a lateral view of the human brain, with various parts marked. Red stars are scattered around the four lobes of the human brain. The frontal area is labeled Components of an episodic memory, and the area above the brain lobe is labeled Hippocampus. The illustration labeled B shows a lateral view of the human brain. It shows the stars that represent episodic memory, linked through the hippocampus. The illustration labeled C shows a lateral view of the human brain. It shows the five stars linked to each other.

standard consolidation theory

The theory that the hippocampus and related medial temporal lobe structures are required for storage and retrieval of recent episodic and semantic memories but not older ones.

Multiple Trace Theory

Other researchers noted that some patients' amnesia does not fit the profile depicted in standard consolidation theory. For example, some patients have severe retrograde memory loss that extends as far back as childhood and is not temporally graded ([Cipolotti et al., 2001](#); [Nadel & Moscovitch, 1997](#)). To account for such extensive retrograde amnesia, Morris Moscovitch and

Lynn Nadel developed [multiple trace theory](#) ([Moscovitch, Cabeza, Winocur, & Nadel, 2016](#); [Nadel & Moscovitch, 1997](#); [Winocur & Moscovitch, 2011](#)). According to multiple trace theory, when an event is experienced, it can be stored as an episodic memory by an ensemble of neurons in the hippocampus and in neocortex (similar to [Figure 7.18B](#)). Each time that memory is retrieved, the retrieval itself becomes a new episodic memory. Thus, an old and often-recalled event can have multiple “memory traces” in the brain.

multiple trace theory

The theory that episodic (and possibly semantic) memories are encoded by an ensemble of hippocampal and cortical neurons and that both the hippocampus and cortex are normally involved in storing and retrieving even very old memories.

Over time, after many such retrievals, the episodic content becomes blurred, but the rich semantic content remains, stored in the cortex independent of the hippocampus, as in [Figure 7.18C](#). Any persisting episodic memory for specific details of the spatial and temporal context would remain dependent on the hippocampus, as in [Figure 7.18B](#). In effect, the hippocampus would serve as an “index,” helping to link together all the semantic elements into one memory of a specific episode tagged with information regarding the time and place where it occurred ([Eichenbaum et al., 2007](#)).

According to this view, individuals with hippocampal damage would effectively lose *all* their episodic memories ([Nadel, Samsonovich, Ryan, & Moscovitch, 2000](#)). Such individuals might be able to rehearse a piece of autobiographical information so many times that it becomes a semantic memory, but this is a far cry from how healthy people normally recall episodic memories. It would be equivalent to the way a person might claim to “remember” her first birthday because she has heard the family stories so often. She has semantic information about the event and knows it happened to her, but that is not the same thing as remembering the episode firsthand. In short, according to multiple trace theory, individuals with medial temporal lobe damage lose the capacity for “mental time travel”—and with it the capacity for true episodic memory ([Steinvorth, Levine, & Corkin, 2005](#)).

Evidence from Healthy Brains

The debate between proponents of standard consolidation theory and those favoring multiple trace theory continues, and various studies have attempted to test the predictions of each.

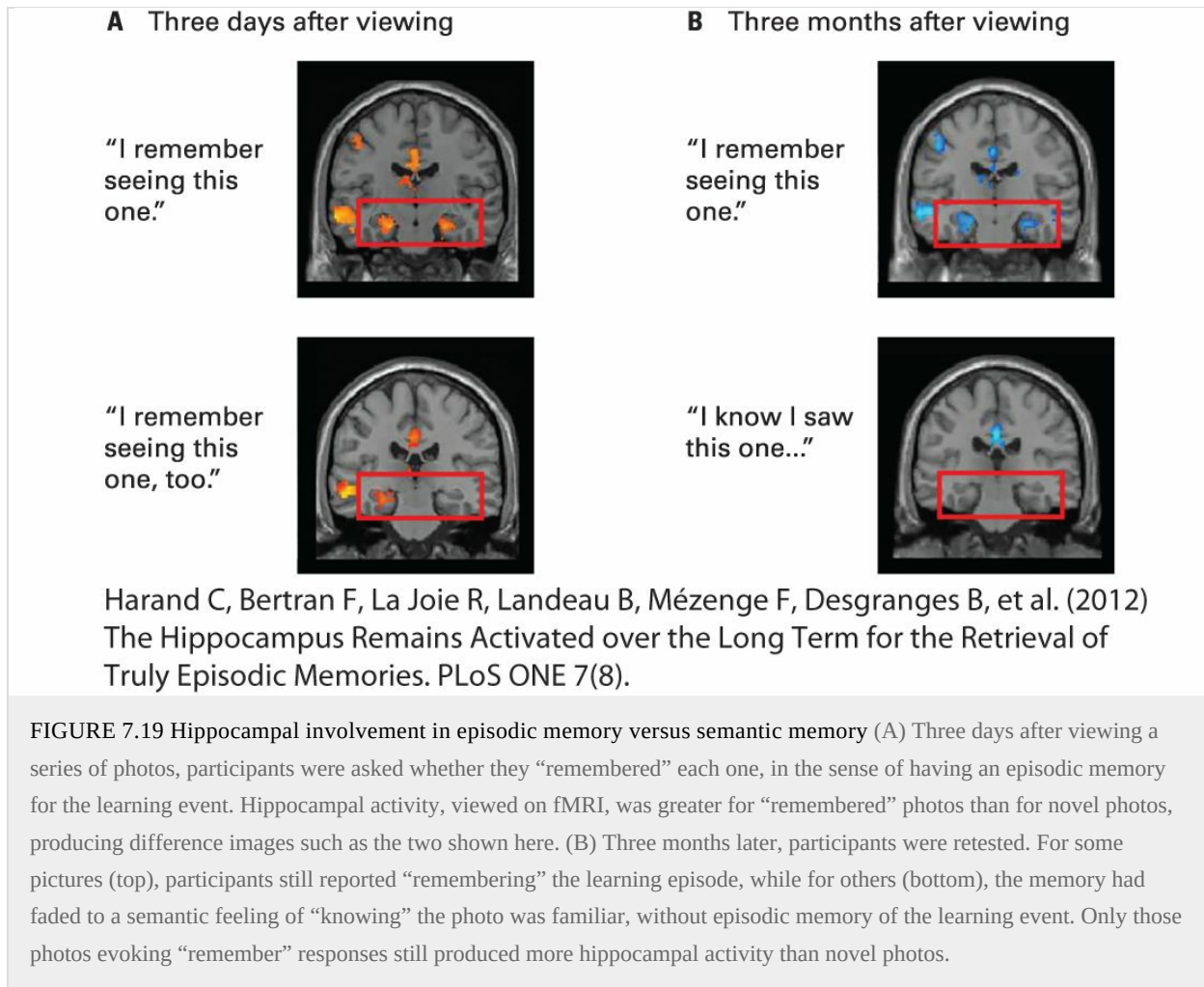
For example, standard consolidation theory predicts that hippocampal activity during memory retrieval should be greatest for recently acquired episodic memories (which are still dependent on the hippocampus, as in [Figure 7.18B](#)) and lowest for very old episodic memories (which should be fully consolidated and independent of the hippocampus, as in [Figure 7.18C](#)). Multiple trace theory predicts that the hippocampus should be equally active during recall of recent and remote episodic memories because the hippocampus is always involved throughout the life of an episodic memory ([Winocur, Moscovitch, & Bontempi, 2010](#)).

To date, an increasing number of studies have found equal hippocampal activation for recent and remote episodic memories ([Bernard et al., 2004](#); [Kapur, Friston, Young, Frith, & Frackowiak, 1995](#); [Maguire, 2001](#)), which is evidence in favor of multiple trace theory. For instance, in one study, when participants were asked to recall specific episodic memories from their personal lives in as much detail as possible, fMRI showed activation of the hippocampus regardless of whether the experience happened 1 week, 1 month, 1 year, or 10 years previously ([Söderlund, Moscovitch, Kumar, Mandic, & Levine, 2012](#)). In other words, there was no evidence that the hippocampus becomes less involved as episodic memories age.

On the other hand, hippocampal activity does seem to decrease over time as the episodic information we “remember” fades into semantic information we merely “know” ([Harand et al., 2012](#); [Viskontas, Carr, Engel, & Knowlton, 2009](#)). In one study, healthy adults viewed a series of photos; 3 days later, while in the fMRI scanner, participants were shown a series of photos. They were asked whether they had seen each photo previously and, if so, whether they had a specific episodic memory of seeing it (“I remember seeing this photo because, when I first saw it, it made me think about my trip to Mexico”) or merely had a general feeling of “knowing” they had seen the picture before, without a memory for the specific details of the learning episode ([Harand et al., 2012](#)).

Unsurprisingly, the hippocampus was more active for photos the participants specifically “remembered” ([Figure 7.19A](#)) than for novel photos. When the same participants were brought back to the lab 3 months later, they still “remembered” some of the photos, and for those the hippocampal activity remained strong. But for other photos, the episodic memory had faded to a semantic memory of “knowing” they had seen the photo, without specific memory of the viewing event. The hippocampus was still active for “remembered” photos but not for “known”

photos (Figure 7.19B)—consistent with the idea that semantic memories can eventually become independent of the hippocampus as the specific episodic details fade.



Description

In illustration A, four magnetic resonance images representing, 3 days after encoding, similar retrieval-related neural network, in the descending order, depicting RR-CR, are shown in the left with a rectangular scale on the left showing T-value ranging from 0 to 10. The first three images are shown in a sagittal view, and the fourth image is shown in the cross-sectional view of a human brain with the left and right hemispheres clearly distinguished, and labeled L and R. The four scans have the following values: first sagittal view: x equals negative 46. second sagittal view: x equals negative 20. third sagittal view: x equals negative 6, and fourth cross-sectional view: y equals negative 14. In illustration A, four magnetic resonance images representing, 3 days after encoding, in the descending order, depicting RR-CR, are shown in the right with a rectangular scale on the left showing T-value ranging from 0 to 10. The first three images are shown in a sagittal view, and the fourth image is shown in the cross-sectional view of a human brain with the left and right hemispheres clearly distinguished, and labeled L and R. The four scans have the following values: first sagittal view: x equals negative 48. second sagittal view: x equals negative 20. third sagittal view: x equals negative 10, and fourth cross-sectional view: y equals negative 18. In illustration B, four magnetic resonance images

representing, 3 months after encoding, activation of the hippocampus only for RR memories, in the descending order, depicting RR-CR, are shown in the left with a rectangular scale on the left showing T-value ranging from 0 to 12. The first three images are shown in a sagittal view, and the fourth image is shown in the cross-sectional view of a human brain with the left and right hemispheres clearly distinguished, and labeled L and R. The four scans have the following values: first sagittal view: x equals negative 52. second sagittal view: x equals negative 24. third sagittal view: x equals negative 6, and fourth cross-sectional view: y equals negative 26.

In illustration B, four magnetic resonance images representing, 3 months after encoding, activation of the hippocampus only for RR memories, in the descending order, depicting RR-CR, are shown in the right with a rectangular scale on the left showing T-value ranging from 0 to 8. The first three images are shown in a sagittal view, and the fourth image is shown in the cross-sectional view of a human brain with the left and right hemispheres clearly distinguished, and labeled L and R. The four scans have the following values: first sagittal view: x equals negative 46. second sagittal view: x equals negative 26. third sagittal view: x equals negative 2, and fourth cross-sectional view: y equals negative 26.

The Role of Sleep

How might a new memory, initially encoded in the hippocampus, eventually get stored in the cortex? A large body of research now suggests that sleep plays an important role ([Diekelmann & Born, 2010](#); [Marshall & Born, 2007](#)). Specifically, humans show a slight but reliable improvement on declarative memory tasks (as well as other forms of learning) if they sleep between learning and retrieval (for review, see [Diekelmann & Born, 2010](#)). Conversely, sleep deprivation negatively affects attention, learning, and memory in humans, rats, birds, and fruit flies (for review, see [Cirelli & Tononi, 2008](#)). So what goes on while we sleep?

Over the course of a night, the human brain goes through several sleep stages, each of which is defined by specific patterns of brain activity (detectable on EEG) and metabolism. These stages include **rapid eye movement (REM) sleep**, during which the eyes move back and forth rapidly under closed lids, and **non-REM sleep**, which includes both light sleep and deeper **slow-wave sleep (SWS)**, so called because of large, slow oscillations in the brain that are highly synchronized over widespread brain areas. Each night, we cycle from light sleep to SWS to REM and back, about every 90 minutes, or about four to six times during a good night's sleep. Similar sleep stages and sleep cycles are thought to occur in most other species, from primates and birds to lizards and cockroaches, although different species vary widely in the frequency and length of these stages (for review, see [Cirelli & Tononi, 2008](#)).

rapid eye movement (REM) sleep

A phase of sleep in which the eyes move rapidly under closed lids.

non-REM sleep

A category of sleep that includes both light sleep and slow-wave sleep (SWS).

slow-wave sleep (SWS)

A phase of sleep characterized by large, slow oscillations in the brain that are highly synchronized over wide brain areas.

Something very interesting happens in the hippocampus during slow-wave sleep. Networks of neurons that were active during the day become active again, seeming to rapidly and spontaneously “replay” activity patterns that they experienced during the day ([Lee & Wilson, 2002](#); [Nádasy, Hirase, Czurkó, Csicsvari, & Buzsáki, 1999](#); [Wilson & McNaughton, 1994](#)). As an example, consider the behavior of “place cells” in a rat hippocampus, a category of neurons described in [Chapter 3](#) that fire preferentially when the rat is in a particular location ([Figure 3.16](#)). During the day, a rat learns to run along a simple track, from left to right, as illustrated in [Figure 7.20A](#). Place cells in the rat’s hippocampus fire in sequence as the rat moves from location to location along the track. Some neurons (coded in purple in [Figure 7.20A](#)) fire when the rat starts at the left end of the track; others (coded in green in [Figure 7.20A](#)) fire when the rat reaches the middle of the track; and still others (coded in yellow and red in [Figure 7.20A](#)) fire as the rat reaches the right end of the track.

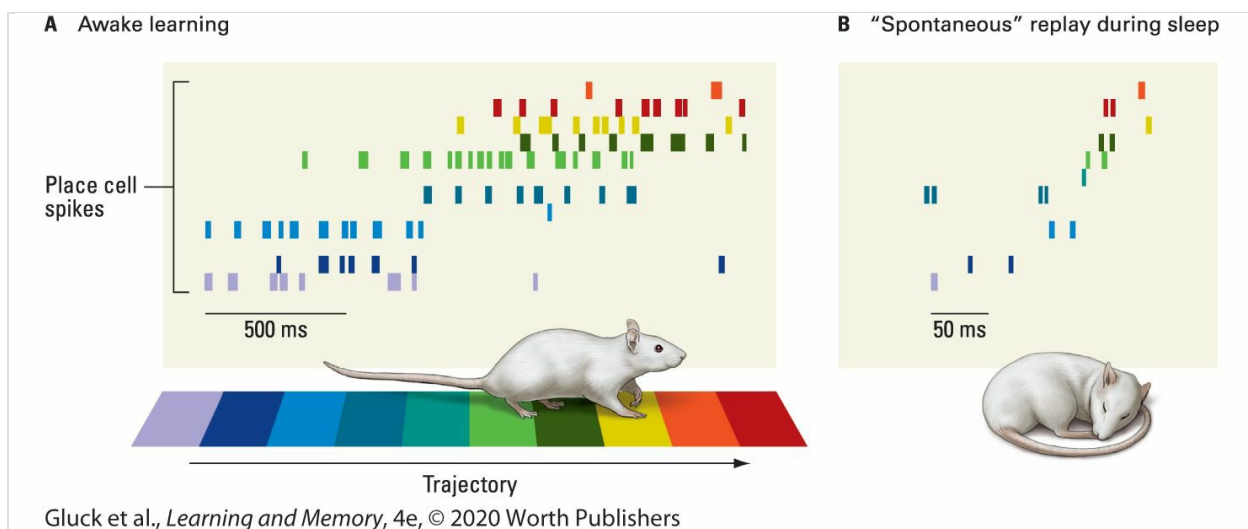


FIGURE 7.20 Spontaneous memory reactivation during sleep (A) During the day, rats learn a task such as running along a track; the track is shown here in a range of colors, from purple (near the beginning) to red (near the end). While the rat runs along the track, researchers record individual hippocampal neurons. Hypothetical responses from 10 neurons are shown, with each neuron coded in different colors. Some (colored purple and blue) fire preferentially when the rat is near the beginning of the track, others (dark and light green) fire preferentially when the rat is in the middle of the track, and still others (yellow and red) fire preferentially when the rat nears the end of the track. (B) That night, during non-REM sleep, the same neurons become active in the same order as during the learning episode—“purple” first, then “blue” and “green,” and finally “yellow” and “red”—as if the rat’s hippocampal neurons were rapidly “replaying” the events of the previous day.

This neuronal reactivation is considered “spontaneous” because it seems to arise from within the brain rather than being evoked by outside stimulation.

Information from Figure 1c, d of [Chen & Wilson, 2017](#).

Description

Illustration A, titled awake learning, shows a track of ten colors, labeled trajectory, of which first and second are shades of red, third and fourth are shades of yellow, fifth and sixth are shades of green, seventh and eighth are shades of blue, ninth and tenth are shades of purple. A rat is shown moving over track is over the shades of blue. Hypothetical hippocampal neuron responses are shown above the track, in which the responses are shown in 500 ms and as boxes of when the rat moves over the respective color. Illustration B, titled spontaneous replay during sleep, shows a rat sleeping and their hypothetical hippocampal neuron responses are shown above, in which the responses are shown in 50 ms and as boxes.

Later, during slow-wave sleep, those same neurons fire—in the same order ([Figure 7.20B](#)); this hippocampal activity while the rat sleeps is synchronized with similar “replay” among neurons in the neocortex that were also active during the daytime learning ([Ji & Wilson, 2007](#); [Sirota, Csicsvari, Buhl, & Buzsáki, 2003](#)). Similar memory reactivation has been observed in other species, and fMRI provides evidence from humans that the same hippocampal areas seen to be activated during the day while participants learned new information are also reactivated during slow-wave sleep that night ([Peigneux et al., 2004](#)).

Many researchers have suggested that such “rapid replay” during sleep is an important mechanism for memory consolidation ([Chen & Wilson, 2017](#); [Diekelmann & Born, 2010](#); [Nádasdy et al., 1999](#); [Wilson & McNaughton, 1994](#)). During the day, we experience many new events and encounter much new information—some of it relevant (the name of a new friend) and some of it irrelevant (what color socks we are wearing). All day long, this incoming information is rapidly encoded in the hippocampus, where it can be stored quickly without disrupting existing cortical networks. Later, during slow-wave sleep, important information is “replayed” in the hippocampus, which in turn reactivates the corresponding memory traces in the cortex. This cortical reactivation allows the cortical networks encoding these memories to be strengthened—but gradually (a little strengthening at each replay)—so that they can be integrated with (but not disrupt) existing memories. In this sense, memory replay during sleep helps us incorporate new declarative memory into our knowledge base, thereby helping make sense of the day’s events. Over time, as the cortical memory is strengthened more and more, the hippocampal replay may become rarer and less important (at least for semantic memories).

It is less clear what role REM sleep plays in consolidating declarative memories. Some researchers believe that declarative memories are first reactivated during slow-wave sleep and then strengthened during subsequent bouts of REM sleep ([Diekelmann & Born, 2010](#)). Because REM sleep is the portion of the sleep cycle most often associated with dreaming, some scientists suggest that dreams may reflect the fragments of memories that are being reactivated and reprocessed while we sleep ([Nielsen & Stenstrom, 2005](#); [Paller & Voss, 2004](#)). Other researchers believe that slow-wave sleep is important for consolidating declarative memories, while REM sleep is more important for consolidating nondeclarative memories, such as the skill learning discussed in [Chapter 8](#) ([Marshall & Born, 2007](#)).

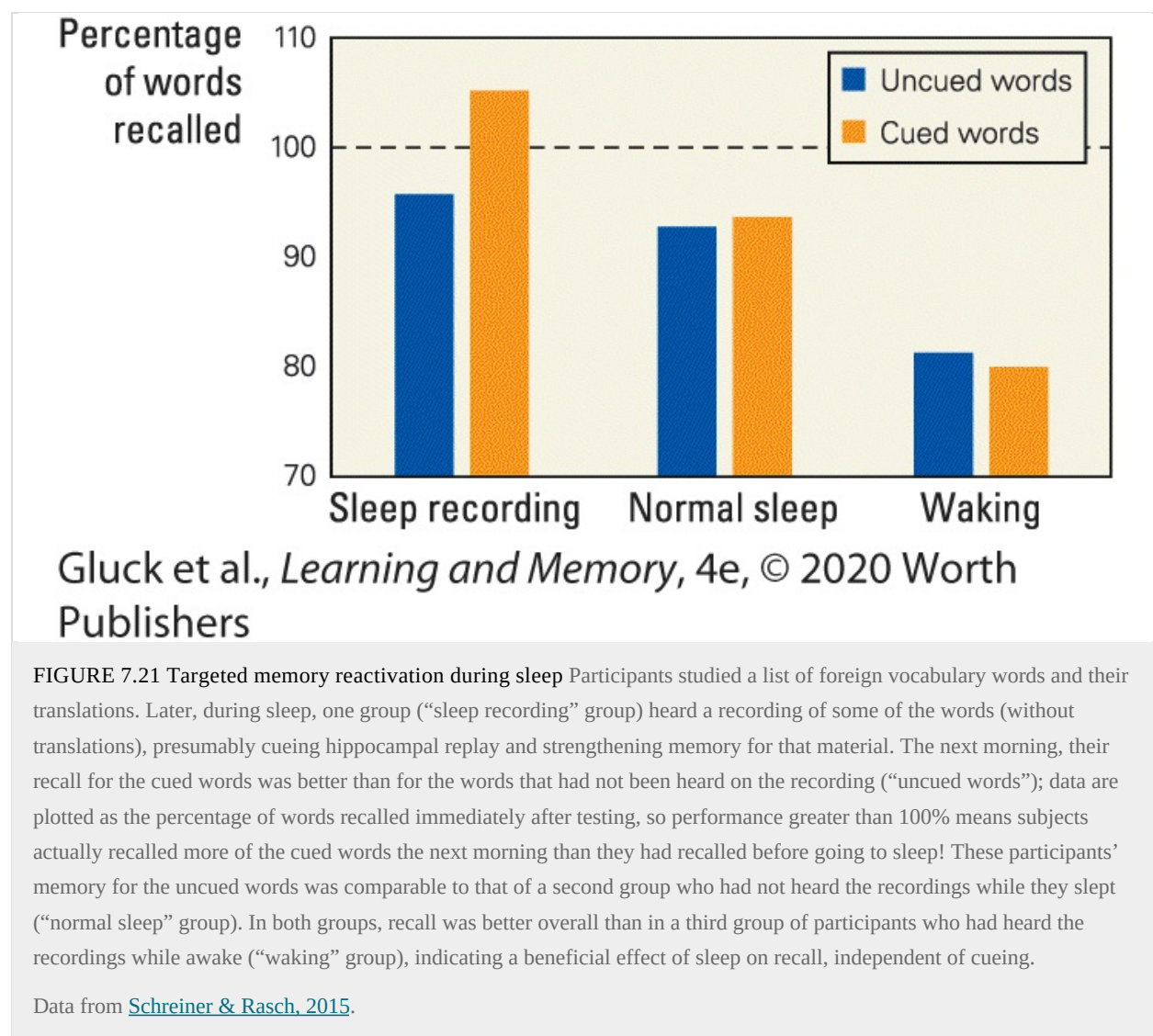
Learning While You Sleep

If sleep indeed plays an important role in memory, then you might wonder whether sleep can be exploited to improve memory. While there are a number of commercial products based on the premise that you can learn new information (such as a foreign language or quantum physics) by listening to audio recordings while you sleep, in fact there is very little scientific evidence to support this idea.

But what about improving memory for material that was already encoded while you were awake? Here there is intriguing evidence from the technique known as *targeted memory reactivation* ([Paller, 2017](#); [Schouten, Pereira, Tops, & Louzada, 2017](#)). In one study, rats learned a task in which auditory cues indicated which of two locations currently contained a reward ([Bendor & Wilson, 2012](#)); during learning, hippocampal neurons showed activation patterns similar to those you saw in [Figure 7.20A](#), indicating the animal's position in space. Later, those same auditory cues were replayed periodically while the rats were in slow-wave sleep—and the cues triggered rapid “replay” of the same activation patterns, in a manner similar to the “spontaneously evoked” replay of [Figure 7.20B](#). Other sounds did not evoke these activation patterns in the hippocampus, nor did the cues themselves when played while the animal was awake or in REM sleep.

Similar effects may occur in humans. In one experiment, a group of participants were given a list of foreign vocabulary words to study ([Schreiner & Rasch, 2015](#)). Later, while the participants slept, a recording of the foreign words (without their translations) was played—very softly, so as not to wake or disturb the sleepers. Presumably, the recording would trigger (or “cue”)

hippocampal replay of the memories, which in turn would facilitate reactivation and strengthening of the memory for the words (and their associated meanings) in cortex. As shown in [Figure 7.21](#), the method worked. The next morning, participants who had heard the recordings during sleep (“sleep recording” group) recalled the cued words and meanings better than participants who had slept without hearing the recording (“normal sleep” group) or participants who had remained awake and heard the same recording (“waking” group). These results also show the general benefits of sleep: even for vocabulary words that had previously been studied but had not been included in the recording (“uncued words” in [Figure 7.21](#)), learning was better in both groups that had slept than it was in the group that had remained awake.



Description

The X-axis is labeled Percentage of words recalled, and Y-axis labeled, Sleep recording, Normal sleep, and Waking. The data of the bar graph are as follows. Under sleep recording, uncued words: 95. cued words, 105. Under Normal sleep, uncued words, 91. cued words, 92. Under Waking, uncued words, 80. cued words, 78. A dashed horizontal line starts from 100 on the vertical axis.

While much remains to be understood about targeted memory reactivation, proponents suggest that targeted reactivation of recently acquired memories during sleep could have important applications, such as helping to speed recovery after a neurological injury, or as a supplement to daily therapy in a mental health setting ([Schouten et al., 2017](#)). And what about studying for your upcoming exam? You will still need to study to encode the information, but do not stay up all night cramming, like the “waking” group in [Figure 7.21](#). Instead, following your study session with a good night’s sleep may help you consolidate memories that you can later retrieve during the exam. And playing that vocabulary list very softly while you sleep might not hurt either.

Interim Summary

- Semantic memories seem to be stored in the cortex; to some extent, specific kinds of semantic information are stored in the cortical areas devoted to processing the corresponding kind of information (visual, auditory, etc.).
- Some research suggests that specific categories of semantic information are stored in specific groups or networks of neurons. Modern techniques have enabled us to view patterns of activity in those networks.
- The hippocampal region lies in the medial temporal lobe in humans and other primates. Patients with bilateral hippocampal-region damage show severe anterograde amnesia (loss of ability to encode new episodic and semantic memories); they typically also show some degree of retrograde amnesia (disruption of old memories acquired before the damage).
- Some researchers propose that the hippocampus is required for new episodic memory formation but that other areas of the medial temporal lobes are sufficient to store new semantic memories; other researchers hold that the hippocampus is necessary for both episodic and semantic memory formation.
- The frontal cortex is important for determining which new memories are stored or forgotten, as well as for metamemory and remembering the source of information. The basal forebrain may help guide how the hippocampus stores new information.
- Standard consolidation theory states that older memories are stored in cortex and eventually become independent of the hippocampus, but multiple trace theory argues that episodic memories always require both the cortex and the hippocampus.

- During slow-wave sleep, neurons in the hippocampus and cortex “replay” in the same sequences in which they fired during learning. This is thought to support the strengthening and integration of long-term memories in the cortex.

7.3 Clinical Perspectives

Patients such as H.M. and E.P. have permanent memory loss caused by identifiable brain damage. E.P.'s amnesia, for example, can be traced directly to medial temporal lobe damage dating to his bout with encephalitis, and H.M.'s amnesia stems from his brain surgery. Once the medial temporal lobes are damaged or destroyed, the lost memories cannot be recovered.

In other cases, however, the memory dysfunction may not be permanent. You have already learned about one kind of “temporary amnesia” in patients who undergo electroconvulsive shock therapy. These patients typically experience anterograde amnesia for the events of the ECT session and retrograde amnesia for events that happened a short time before the session. But their memory machinery is not permanently disabled. After a few hours, their brains are again able to encode new memories and retrieve old ones. (The [“Learning and Memory in Everyday Life”](#) box reviews post-concussive amnesia, which is often temporary but sometimes has longer-lasting effects.) This section reviews two more kinds of amnesia that do not involve permanent disruption of the ability to store memories: transient global amnesia and functional amnesia.

LEARNING AND MEMORY IN EVERYDAY LIFE

The Cost of Concussion

Mild traumatic brain injury (mTBI), also known as concussion, may affect more than 1.7 million people per year in the United States alone ([Carroll, Cassidy, Holm, Kraus, & Coronado, 2004](#)). Typical causes of mTBI are falls, car or bike accidents, and sports injuries; military personnel are at high risk due to exposure to blast waves from nearby explosions ([Langlois, Rutland-Brown, & Wald, 2006](#)).

One of the defining symptoms of mTBI is posttraumatic amnesia, which can include both retrograde and anterograde memory loss. For example, a football player who sustains a concussion may not remember the plays leading up to his on-field collision or the trainers who helped him off the field afterward. One study of young patients (aged 10 to 18 years) with sports-related concussion found that about a quarter reported amnesia for the events surrounding the injury ([Register-Mihalik, De Maio, Tibbo-Valeriotte, & Wooten, 2015](#)). Until fairly recently, it was assumed that the memory loss, like that following ECT, is temporary, such that in the days after the injury, as the other symptoms of concussion (e.g., headache, blurred vision) resolve, so too does the amnesia, with only the memories for the minutes or hours immediately preceding and after the injury permanently lost—forming a small temporal “hole” in the patient’s memory.

But newer research has shown that memory effects may linger much longer. One study examined retired athletes who had sustained a sports-related concussion more than 30 years previously, compared to same-aged former athletes who had never sustained concussions ([De Beaumont et al., 2009](#)). Both groups were given the picture memory task shown in [Figure 7.13A](#). The members of the non-concussed group performed normally for their age group (similar to the healthy controls in [Figure 7.13B](#)). The members of the concussed group performed normally on the copying portion of the task but were impaired in their ability to draw the figure from memory. This pattern is similar to, although much less severe than, that shown by

amnesic patient E.P. in [Figure 7.13C](#).

Worse, individuals who have suffered one concussion are vulnerable to poorer outcomes if they suffer a second concussion, particularly if the second injury occurs before the symptoms from the first one have completely resolved ([Ling, Hardy, & Zetterberg, 2015](#)). Individuals who sustain repeated mTBI, such as some athletes who play contact sports, are also at increased risk for cognitive decline and dementia late in life ([Danshevar et al., 2011](#)). American football is one such sport, and it has come under intense scrutiny in recent years following the deaths of several high school football players—including three in one week in October 2014 ([Carver, 2014](#))—and a lawsuit against the National Football League (NFL) by former players who claimed the league did not adequately disclose the risks of concussion ([Martin, 2013](#)).

Meanwhile, a study involving autopsies on the brains of 111 former NFL players found that all but 1 showed signs of *chronic traumatic encephalopathy*, or CTE ([Mez et al., 2017](#)), a neurodegenerative disease caused by repetitive head trauma that can also cause depression, memory loss, and poor judgment ([Asken, Sullan, DeKosky, Jaffee, & Bauer, 2017](#)). Indeed, 18 of the 111 players had committed suicide, and another 7 had died from unintentional drug overdose.

As a result of the emerging findings, many school athletic programs now require protective head gear; have strict rules governing return to play for student athletes who sustain concussions; and even prohibit game maneuvers such as head hits in hockey, headers in soccer, and head-down tackling in football ([Ling et al., 2015](#)). Meanwhile, in 2013, the NFL settled its lawsuit, agreeing to pay \$675 million to retired players suffering from long-term effects of concussion and \$10 million to fund brain injury research and educational programs ([Martin, 2013](#)).

Transient Global Amnesia

As its name suggests, **transient global amnesia (TGA)** is a transient, or temporary, disruption of memory not due to known causes such as head injury or epilepsy ([Brand & Markowitsch, 2004](#); [Kritchevsky, Squire, & Zouzonis, 1988](#); [Shekhar, 2008](#)). Underlying causes are enigmatic, but about a third of patients undergo an apparent precipitating event such as vigorous exercise or emotional stress that might have affected the flow of blood to the brain ([Hodges & Warlow, 1990](#)). Typically, TGA starts suddenly, persists for several hours, and then gradually dissipates over the course of a day or so. During the amnesic episode, the patient shows severe anterograde amnesia, along with some degree of retrograde amnesia for events that occurred within the preceding decade or so ([Kritchevsky & Squire, 1989](#); [Kritchevsky et al., 1988](#)).

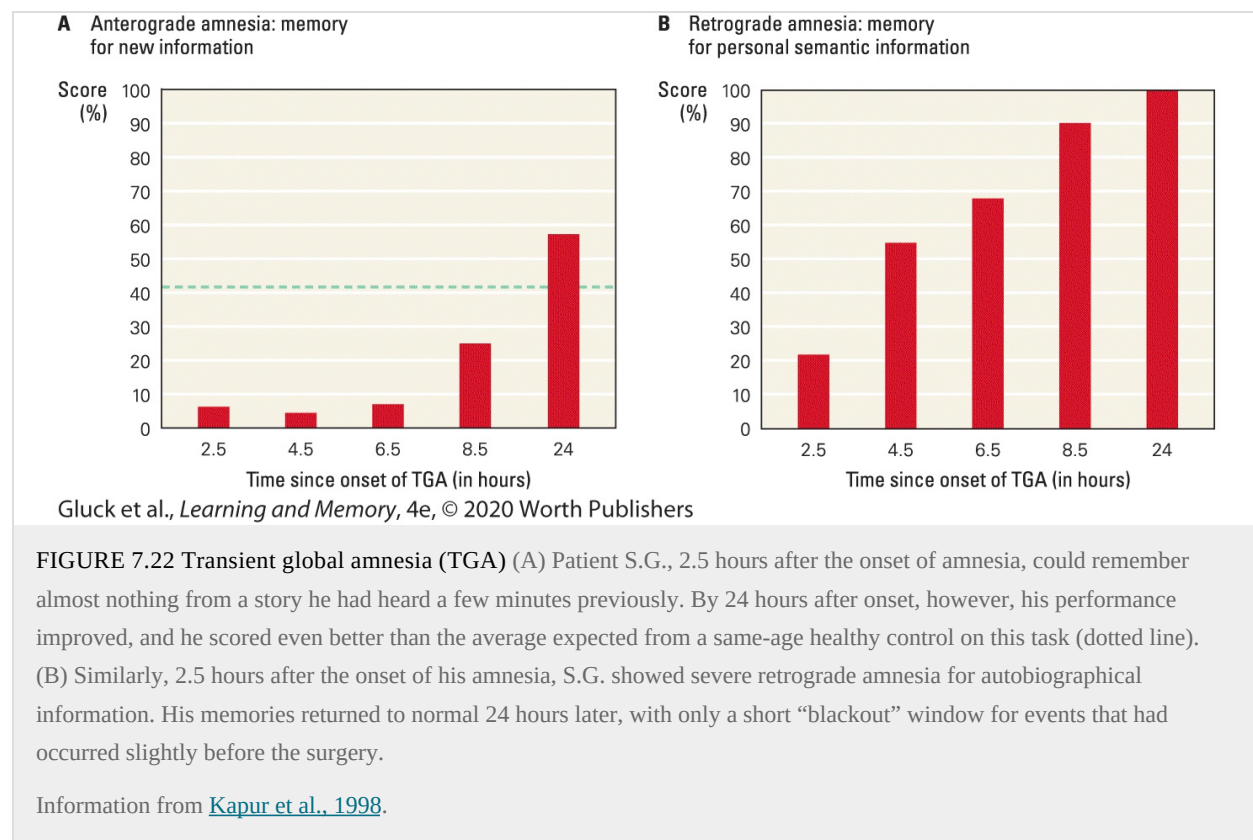
transient global amnesia (TGA)

A transient, or temporary, disruption of memory typically including elements of both anterograde and retrograde amnesia.

Transient global amnesia is one of the more common forms of amnesia; annual incidence has been estimated at about 5 cases per 100,000 people per year in the United States ([Miller, Petersen, Metter, Millikan, & Yanagihara, 1987](#)). Despite the prevalence, TGA is difficult to study because it does not usually last long. But there are a few well-documented cases in the

literature. One such case involves S.G., a 38-year-old man who underwent brain surgery ([Kapur, Millar, Abbott, & Carter, 1998](#)). The surgery seemed to go smoothly, but there may have been some complication that temporarily reduced blood flow to his brain. When S.G. woke up, he knew his own name but did not remember his occupation, the month, or how long he had been in the hospital.

The onset of S.G.'s amnesia occurred around 12:30 p.m. Memory researchers rushed to the scene, and S.G. agreed to an extensive battery of testing. At about 3 p.m., S.G. showed profound anterograde amnesia: he would listen to a short story, but a few minutes later he could recall only a few words of it ([Figure 7.22A](#)). S.G. also showed retrograde amnesia. Given a questionnaire about jobs he had held, places he had lived, and other personal semantic information, he could provide only a few answers ([Figure 7.22B](#)). (The correct answers were verified by S.G.'s fiancée.) S.G.'s memory was similarly poor for recent public events, although it was better for events that had happened at least a few decades prior.



Description

The graph labeled A shows Anterograde amnesia: memory for new information. The X-axis shows Score (Percent), and

Y-axis shows Time since onset of TGA (in hours). A dotted line lies horizontally at point 40. The data in the graph are as follows. At 2.5 hours, 4 percent. At 4.5 hours, 3 percent. At 6.5 hours, 7 percent. At 8.5 hours, 25 percent. At 24 hours, 58 percent.

The graph labeled B shows Retrograde amnesia: memory for personal semantic information. The X-axis shows Score (Percent), and Y-axis shows Time since onset of TGA (in hours). The data in the graph are as follows. At 2.5 hours, 21 percent. At 4.5 hours, 53 percent. At 6.5 hours, 67 percent. At 8.5 hours, 90 percent. At 24 hours, 100 percent.

Researchers continued to test S.G. every few hours through the afternoon and evening; gradually, his anterograde and retrograde amnesia lessened. By noon of the next day, 24 hours after the onset of amnesia, S.G.'s memory had returned to normal, except for a slight retrograde amnesia for events that had occurred shortly before the surgery. The TGA was over; S.G.'s brain seemed to be back in working order.

Why might TGA occur? Like S.G., most individuals with TGA appear to have experienced a precipitating event, such as vigorous exercise, dramatic temperature changes (such as swimming in cold water), or extreme emotional stress ([Quinette et al., 2006](#)). (In a few cases, migraine headaches are implicated.) These events might have caused temporary interruption of blood flow to the brain. As in ECT, the resulting disruption in neuronal activity might completely prevent formation of memories for ongoing events for the duration of the TGA but might only temporarily limit access to fully consolidated older memories. When the blood flow resumes, so does the brain function.

Consistent with this idea, studies using neuroimaging methods such as positron emission tomography (PET) and magnetic resonance imaging (MRI) suggest that many individuals experiencing an episode of TGA show small abnormalities in the hippocampus ([Gonzalez-Martinez, Comte, de Verbizier, & Carlander, 2010](#); [Yang, Kim, Kim, Kwak, & Han, 2009](#)). In some cases, these abnormalities are no longer detectable on follow-up imaging 2 months later, after the TGA has resolved ([Alberici et al., 2008](#); [Della Marca et al., 2010](#)). While this may not explain all cases of TGA, it suggests that the temporary disruption of memory storage and recall may reflect temporary disruption of the hippocampus.

Functional Amnesia

In the 2002 film *The Bourne Identity*, Jason Bourne (played by Matt Damon) awakes on a fishing

boat with no memory of his own name, his past, or why he has the number of a Swiss bank account implanted in his hip. Throughout the movie, Bourne struggles to regain his memories while staying one step ahead of the CIA agents who are trying to kill him. Such memory loss has formed the basis of countless books, movies, and plays—from Alfred Hitchcock’s 1945 classic *Spellbound* to 2014’s *The Maze Runner*, based on James Dashner’s novel in which a 16-year-old hero wakes up trapped in a maze with no memory of his prior life, to an episode of *The Simpsons* in which Marge develops amnesia and forgets all about her dysfunctional animated family.

Such memory loss can also happen in real life, although it is far rarer than Hollywood might lead you to believe. **Functional amnesia** (sometimes called *psychogenic amnesia*) is a sudden, massive retrograde memory loss that seems to result from psychological causes, in contrast to *organic amnesia*, which arises from obvious physical causes, such as brain injury ([Kritchevsky, Chang, & Squire, 2004](#); [Schacter & Kihlstrom, 1989](#)). One type of functional amnesia is *dissociative amnesia*, in which patients lose memory of a specific traumatic event; an even more dramatic type is *dissociative fugue*, in which patients lose all memory of their identity. Dissociative fugue is extraordinarily rare. Moreover, some individuals who claim to have lost their memory will later admit to faking functional amnesia in order to avoid dealing with a difficult situation such as a crime or a relationship problem ([Jenkins, Kapur, & Kopelman, 2009](#)). Some cases, however, do seem to involve genuine memory loss.

functional amnesia

A sudden retrograde memory loss that seems to result from psychological causes rather than physical causes such as brain injury; also called *psychogenic amnesia*.

Daniel Schacter records one case of functional amnesia involving a 21-year-old man, P.N., who was admitted to the hospital complaining of back pains ([Schacter, Wang, Tulving, & Freedman, 1982](#)). When questioned about his identity, P.N. could not remember his name or anything about his past except that he had once been given the nickname “Lumberjack.” Schacter later concluded that P.N. had developed functional amnesia following the death of his grandfather, to whom P.N. had been close. P.N.’s extreme grief was the psychological trauma that triggered the memory loss.

In contrast to P.N.’s severe retrograde amnesia for autobiographical events, his semantic memories were intact: both his language functions and his knowledge about the world seemed normal. However, P.N. also displayed anterograde amnesia, having difficulty remembering new

information for more than a few minutes at a time. The functional amnesia persisted for about a week, until P.N. happened to watch a television show that included a funeral scene; as if by magic, lost memories came flooding back, and P.N. recalled his own identity and history. Only the memories of events that occurred during the previous week were permanently lost, possibly because his anterograde amnesia had prevented those memories from being successfully stored in the first place.

Not all cases of functional amnesia resolve so neatly. Mark Kritchevsky and his colleagues studied 10 individuals with functional amnesia; none were able to report their names or personal histories ([Kritchevsky et al., 2004](#)). One of these patients later admitted to feigning his amnesia, and the researchers suspected a second may have been feigning, too. But of the 8 patients whose amnesia seemed genuine, only 1 fully recovered all the lost memories. Another case study involving a woman who lost the memories of her identity and the past 33 years of her life reported that her memories had not returned even after 11 years ([Helmes, Brown, & Elliott, 2015](#)). Currently, there is no way to predict when or whether particular patients will recover their memory.

Given that patients with functional amnesia have no known brain damage, what could cause this syndrome? Functional imaging may provide some clues. In one study using PET, an individual with functional amnesia had abnormal activity patterns in areas of the medial temporal lobes; these abnormalities disappeared when the amnesia resolved ([Markowitsch et al., 1998](#)). This suggests that functional amnesia may result from a (possibly temporary) malfunction of the brain areas normally involved in episodic memory storage and retrieval. Another study using fMRI showed that when two people with functional amnesia “failed to recognize” individuals with whom they were actually acquainted, there was increased activity in the prefrontal cortex and decreased activity in the hippocampus ([Kikuchi et al., 2010](#)). This is the same basic pattern observed during directed forgetting in healthy adults (see [Figure 7.16](#)), which suggests that—consciously or unconsciously—the patients may have been suppressing recall. Interestingly, when one patient’s amnesia resolved, the abnormal pattern of prefrontal activation disappeared.

Together, these imaging results suggest that functional amnesia may reflect dysfunction of the same brain regions that are damaged in organic amnesia ([Ouellet, Rouleau, Labrecque, Bernier, & Scherzer, 2008](#)). The major differences would be that, whereas organic amnesia is brought on

by a physiological injury, functional amnesia is brought on by a psychological trauma; thus, because there is no permanent injury to the brain, it may be possible for patients with functional amnesia to recover both their brain function and their memories in a way that would be impossible for patients such as H.M. and E.P.

Test Your Knowledge

Don't Forget Your Amnesias

Amnesia is a general term for memory loss. For each specific type of amnesia listed below, can you remember what kind of information or functioning is lost or disrupted, as well as what kind of trauma or damage might be responsible? ([Answers appear at the end of the chapter.](#))

1. Anterograde amnesia
2. Functional amnesia
3. Retrograde amnesia
4. Transient global amnesia (TGA)

Interim Summary

- Transient global amnesia (TGA) is a temporary disruption of memory that occurs without evidence of head injury or epilepsy; however, there may be precipitating events such as vigorous exercise or stress.
- During a TGA episode, patients typically show severe anterograde amnesia and usually some degree of retrograde amnesia.
- TGA may reflect a temporary disruption of brain function due to interruption of blood to the brain; typically, as the brain recovers, memory returns to normal.
- Functional amnesia is a very rare form of amnesia in which patients lose memory for specific episodes in their lives or even forget their entire identity.
- Functional amnesia seems to result from psychological trauma rather than any physical cause.

Synthesis

[Section 7.3](#) mentioned a few examples of the many movie and television characters who have been depicted as having developed amnesia. Knowing what you now know about memory and amnesia, you should be able to assess the plausibility of these characters—as well as of amnesia portrayals in other movies, TV shows, and books.

The vast majority of them, like Jason Bourne in *The Bourne Identity*, are afflicted with functional amnesia—usually a complete loss of identity caused by psychological trauma rather than physical brain damage. These characters typically maintain their semantic memory, as well as their skills. This profile is roughly similar to that observed in real-world patients such as P.N., whose functional amnesia stripped him of his personal history, though his general knowledge about the world seemed intact.

Jason Bourne (like the protagonists of many other amnesia stories) spends much of the movie trying to solve the mystery of what triggered his amnesia, as well as trying to recover his lost identity. In this sense, *The Bourne Identity* and other “lost identity” stories share many features with real-world cases of functional amnesia. Where these stories depart from reality is mainly in their resolution: in many a TV drama, the heroine who has lost her memory following a blow to the head needs nothing more than a second blow to bring the memories flooding back. In the real world, head injuries do not cure functional amnesia. Instead, as you also read, even mild head injuries can cause lasting memory problems. This was the subject of the 2015 biopic *Concussion* that starred Will Smith as Dr. Bennet Omalu, the real-world pathologist who confronted the NFL over his findings of neurodegeneration in the brains of former professional football players.

Organic amnesia is a much less common plot device than functional amnesia. Exceptions include Christopher Nolan’s 2000 film *Memento*, a psychological thriller featuring a protagonist who suffers from anterograde amnesia, as well as Pixar’s 2003 *Finding Nemo* and the 2016 sequel *Finding Dory*, which feature an animated fish (voiced by Ellen DeGeneres) with the same disorder. In both these cases, the afflicted characters are intelligent and motivated individuals who are unable to acquire new episodic or semantic memory; anything they learn or do is forgotten within a few minutes. *Memento*’s protagonist explains that he developed his condition following a head injury that damaged his hippocampus. These fictional characters share many characteristics with real-world amnesic patients such as H.M. and E.P., who developed

anterograde amnesia following medial temporal lobe damage. However, real-world patients such as H.M. and E.P. usually have at least some retrograde amnesia as well—loss of some (and often quite a lot) of their declarative memories from before the brain damage.

Yet another class of memory-related movie plots revolve around false memories. For example, in the sci-fi classic *Total Recall* (starring Arnold Schwarzenegger in 1990; remade in 2012 starring Colin Farrell), the protagonist pays to have false memories of an expensive trip to Mars implanted in his brain—at a fraction of the time and cost that it would take to actually vacation on another planet. In *The Manchurian Candidate* (starring Frank Sinatra in 1962; remade in 2004 starring Denzel Washington), an evil corporation uses brainwashing and hypnotism to implant false memories. These stories are obviously science fiction, but the phenomenon of false memories is science fact: under the right conditions, researchers can implant false memories that feel as rich and real as true memories—so real that research participants will often argue when researchers attempt to explain the hoax. False memories lead to exciting adventures in movies, but they can lead to serious real-world problems, as when eyewitnesses confuse memories of a crime scene with pictures of a suspect aired on the nightly news.

In all of these plots, the drama springs from a universal sense that our episodic and semantic memories—the facts we know and the events we have experienced—make us who we are. It is easy to empathize with a character whose memories have been stripped away because we can imagine the devastating impact.

On a more mundane level, everyone experiences occasional failures of episodic and semantic memory. Some of these failures, such as forgetting where we parked the car or blanking on the name of someone we see unexpectedly at the gym, are just a normal part of everyday life. Some of us have experienced an episode of TGA following a sports injury, have fallen prey to source monitoring errors while writing a term paper, or have experienced judgment-of-learning errors while studying for an exam. Most of the time, our access to episodic and semantic memory is effortless, and our memories are long-lasting and largely accurate—but at the same time, our memory processes may fail more often than we like. Understanding these processes may help us improve their function while giving us—like Jason Bourne and his fellows—a better appreciation of the episodic and semantic memories we do possess.

Chapter 7 Review

Know Your Key Terms

amnesia

anterograde amnesia

association cortex

basal forebrain

confabulation

consolidation period

cued recall

declarative memory

directed forgetting

electroconvulsive shock

encoding specificity effect

episodic memory

explicit memory

false memory

fornix

free recall

frontal cortex

functional amnesia

hippocampus

implicit memory

interference

levels-of-processing effect

medial temporal lobes

metamemory

multiple trace theory

non-REM sleep

nondeclarative memory

proactive interference

rapid eye movement (REM) sleep

recognition

reconsolidation

[retroactive interference](#)
[retrograde amnesia](#)
[Ribot gradient](#)
[semantic memory](#)
[sensory cortex](#)
[slow-wave sleep \(SWS\)](#)
[source monitoring error](#)
[standard consolidation theory](#)
[transfer-appropriate processing effect](#)
[transient global amnesia \(TGA\)](#)

Quiz Yourself

1. The general term for conditions involving severe memory loss is _____. More specifically, loss of the ability to form new declarative memories is called _____, while the loss of previously acquired information is called _____. ([pp. 277, 305, 311](#))
2. Three ways in which memory can be tested are free recall, which entails _____; cued recall, which entails _____; and recognition, which entails _____. Of the three, _____ usually produces the best performance. ([p. 289](#))
3. The “_____ effect” refers to the phenomenon that memory is usually best when the cues available at testing are similar to those at encoding; the “_____ effect” refers to the phenomenon that memory is usually best when information is processed in the same way at encoding and retrieval. ([pp. 287–288](#))
4. In the _____ paradigm, subjects typically show worse memory for items that they have been specifically instructed to forget. ([p. 292](#))
5. Newly acquired episodic and semantic memories are particularly vulnerable during the _____. However, each time a memory is reactivated or recalled, it may again become vulnerable to modification, a process termed _____. ([pp. 296, 298](#))

6. _____ occurs in some forms of amnesia when patients, asked to remember past events, respond with highly detailed but false memories. ([p. 310](#))
7. _____ is memory for facts and information about the world (including personal information); _____ is memory for specific events that occurred at a particular time and place. ([pp. 278–279](#))
8. The _____ is located in the medial temporal lobe of the brain and is connected to subcortical structures such as the _____ by a fiber bundle called the _____. ([p. 309](#))
9. A false memory is _____. ([p. 294](#))
10. Areas of the cerebral cortex involved in processing specific types of information, such as sights and sounds, are called _____; other areas of the cortex, called _____, process information both within and among sensory modalities. ([p. 301](#))
11. The Ribot gradient describes a pattern of memory loss in which _____ memories are more prone to disruption than are _____ memories. ([p. 311](#))
12. _____ is a sudden memory loss that does not seem to result from physical causes such as brain injury. ([p. 321](#))
13. During periods of _____, neurons in the hippocampus “replay” the same activation patterns that they showed during prior learning. ([p. 316](#))
14. Therapy involving _____ is sometimes used to treat severe depression but can severely disrupt newly formed memories. ([p. 297](#))
15. Whereas _____ memory includes information we are aware that we know, _____ memory can exist without the learner’s awareness. ([p. 280](#))
16. Severe and permanent amnesia can be caused by brain damage that includes the _____ or the _____. ([pp. 305, 310](#))
17. _____ refers to a disruption of memory due to overlap with the content of

other memories. In _____, new learning disrupts old (previously stored) information. In _____, old learning interferes with the ability to recall newly learned information. ([p. 293](#))

18. Feeling-of-knowing and judgment-of-learning phenomena are examples of _____, the knowledge of, and ability to think about, our memories. ([pp. 298–299](#))
19. Episodic memory and semantic memory are sometimes grouped together in the category _____, whereas other types of memory, such as skill memory, that are not always consciously accessible or easy to verbalize are sometimes grouped together in the category _____. ([p. 280](#))
20. _____ is a transient or temporary disruption of memory that typically includes both anterograde and retrograde amnesia. ([p. 320](#))
21. Standard consolidation theory states that brain structures including the _____ are required for storage and retrieval of _____ memories but not of _____ memories. In contrast, multiple trace theory suggests that _____. ([pp. 313–314](#))
22. Remembering a fact but thinking you learned it in school when you actually only saw it in a movie is an example of a _____. ([p. 294](#))

Concept Check

1. Suppose you join a club with six members, and you want to remember each member's name for the next meeting. What are three ways, based on the principles described in this chapter, that you can improve the likelihood of remembering the names?
2. A semantic memory is a memory for a fact without memory of the spatial and temporal context in which that fact was learned. How does this differ from source monitoring error?
3. Failures of episodic memory and semantic memory can be annoying, but they serve a

purpose. Why might it be desirable for an organism to be able to forget some information?

4. In adult humans with normal memory function, fMRI shows that the hippocampus is active even for retrieval of very old episodic information ([Ryan et al., 2001](#)). Does this prove that episodic memories always remain at least partially dependent on the hippocampus?
5. Suppose you are working in an emergency room when a man comes in claiming to have forgotten his entire identity. What questions would you ask the friend who drove him to the hospital? What tests might you conduct to find out what is going on?

Answers to Test Your Knowledge

Episodic Memory Versus Semantic Memory

1. The student remembers the meaning of *carpe diem* using semantic memory, and he has no episodic memory of acquiring this information.
2. Here, the student does have an episodic memory, tagged in space and time, of studying the phrase *ne tentes, aut perfice*, but no semantic memory for what the phrase means in English (roughly, “either succeed or don’t bother trying”).
3. Knowing that the student center serves lousy coffee is an example of general knowledge about the world, so it counts as semantic memory. (Knowing how to get to Starbucks from the present location counts as semantic memory, too.) But why does the college senior think that the coffee is better at Starbucks? If he can remember a specific episode in which he went to the student center and had terrible coffee, or visited Starbucks and had a great experience, that would qualify as episodic memory.

Going Retro

1. This is *retroactive interference*. Scott’s new learning (Spanish) reaches backward in time to disrupt old learning (Latin).
2. This is *proactive interference*. Memory for the old name (Carl) is reaching forward in

time to disrupt memory for the new name (Lance).

3. This is *anterograde amnesia*. Even though the event (hospitalization) happened a long time ago, it happened *after* John's brain injury, and the hippocampal damage at that time prevented him from forming a new memory of it.
4. This is *retrograde amnesia*. John's injury reaches back in time to disrupt memory for events that occurred *before* his injury.

Don't Forget Your Amnesias

1. *What is lost or disrupted*: the ability to form new episodic and semantic memories. *Common causes*: damage to the medial temporal lobes (or the basal forebrain).
2. *What is lost or disrupted*: personal (episodic and semantic) memories. *Common causes*: strong psychological trauma but no obvious physiological injury.
3. *What is lost or disrupted*: the ability to retrieve (recall) existing episodic (and possibly semantic) memories. *Common causes*: broad damage to the medial temporal lobes and beyond.
4. *What is lost or disrupted*: anterograde (and possibly retrograde) memory, usually for a day or less. *Common causes*: unknown but possibly a brief interruption of blood flow to the brain.



Chapter 8 Skill Memory

Learning by Doing



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Behavioral Processes

Features of Skill Memories

Encoding New Memories

Learning and Memory in Everyday Life: Are Some Cognitive Skills Easier for Men Than for Women?

Retrieving Existing Memories

When Memory Fails

Brain Substrates

The Basal Ganglia and Skill Learning

Learning and Memory in Everyday Life: Are Video Games Good for the Brain?

Cortical Representations of Skills

The Cerebellum and Timing

Clinical Perspectives

Parkinson's Disease

Human-Machine Interfaces: Learning to Consciously Control Artificial Limbs

DO YOU LIKE TO DANCE AT CONCERTS AND PARTIES? For some, it is a freeing and joyful experience. For others, it is an awkward and tentative one in which a single thought keeps recurring: "Am I doing this right?" Dancing is simple enough in concept: move your body rhythmically while music is playing. What could be easier? Yet it doesn't take long, especially in social situations, to realize that dancing is not an ability that most humans are born with. By the same token, watching the movements of proficient dancers is enough to make you appreciate that dancing well requires some skill.

Successfully dancing with a partner in a social setting depends in part on the setting and the partner, but most young people are savvy enough to know that they need to practice if they want to impress their friends. Practice might consist of dancing in front of a mirror, or watching videos, or playing Dance Dance Revolution. The hope is that these practice sessions will provide an edge when a real opportunity comes along. Practicing by dancing with a partner you trust is a good strategy because you can get feedback about what you are doing wrong. Taking a class might also help.

Some of what you need to learn about dancing can be shared verbally, but a lot of it is easier to demonstrate than to explain. You will not become adept by memorizing lists of rules about how to dance. To become an expert, someone who performs a skill better than most, you need to get in there and dance (a lot); you need to get to the point where the right movements start feeling familiar and natural. Your brain has to store memories of your dancing successes and failures.

These accumulated experiences can change your abilities whether you are aware of it or not.

expert

A person who performs a skill better than most.

This chapter describes how repeated experiences can incrementally enhance the performance of social dancing or any other skill by gradually modifying memories of how the skill can best be executed. Repeated experiences can change not only how well a person performs a skill but also the structure of the brain circuits that are used to perform that skill, including the basal ganglia, the cerebral cortex, and the cerebellum. People with damage to these brain regions can have trouble learning new skills as well as performing skills already learned.

8.1 Behavioral Processes

[Chapter 7](#) deals with memories for events and facts—in other words, information an individual remembers and knows about. Skill memories, in contrast, relate to what one knows how to do.

A [skill](#) is an ability to perform a task that has been honed through experience. The kinds of skills you are probably most aware of are those that athletes demonstrate when they compete or that musicians reveal when they perform. More mundane skills include driving a car, dancing, and drinking out of a glass. These are all examples of [perceptual-motor skills](#), a category that includes learned movement patterns guided by percepts.



Ulf Huett Nilsson/Johner Images/Getty Images

What sorts of memories do you think might help you avoid the fate of this poor soul?

skill

An ability that can improve over time through practice.

perceptual-motor skill

Learned movement patterns guided by sensory inputs.

The kinds of abilities that you can improve through practice go far beyond fancy limb movements and subtle sensory distinctions. How about playing cards, budgeting your money, taking tests, or managing your time? These are all [cognitive skills](#), which require you to solve problems or apply strategies rather than execute physical maneuvers or sharpen your senses ([Ackerman, 2007](#); [Rosenbaum, Carlson, & Gilmore, 2001](#); [van Lehn, 1996](#)). By reading this sentence, you are exercising a cognitive skill that you learned a long time ago. Reading may now seem so effortless that you can hardly recall the challenge of acquiring this complex ability. When you turn a page, highlight a sentence, take notes, or study the contents of this chapter, you are making use of skill memories.

cognitive skill

A skill that requires problem solving or the application of strategies.

Features of Skill Memories

As you will soon discover, skill memories are similar in many respects to episodic and semantic memories. But they also possess some qualities that set them apart ([Table 8.1](#)). Like memories for facts, skill memories are long-lasting and improved by repeated experiences. Unlike memories for events and facts, however, skill memories can't always be verbalized. As described in [Chapter 7](#), psychologists sometimes classify memories that are not easily put into words, including skill memories, as *nondeclarative memories*, or *implicit memories*. Skill memories may be acquired and retrieved without the awareness and feelings of remembering associated with recalling episodic memories. The introduction to [Chapter 7](#) describes a remarkable case of memory loss in a man known as H.M. It was H.M.'s ability to acquire new skill memories, despite his inability to recall recent episodes, that initially led researchers to classify consciously accessible memories (declarative memories, the kind H.M. could not remember) as being fundamentally different from skill memories.

TABLE 8.1 Comparison of skill memories and memories for events and facts

Skill memories	Memories for events and facts
Are difficult to convey except by direct demonstration	Can be communicated flexibly, in different formats
May be acquired without awareness	Have content that is consciously accessible
Require several repetitions	Can be acquired in a single exposure

Skills can be learned in many ways, but the most common methods involve practice, instruction, and observing others as they perform the skills. [Chapter 5](#) describes one technique, *operant conditioning*, that is commonly used to train animals to perform skills. Pressing a lever, pecking a key, or rolling over on command might seem simple compared to learning to read or play chess, but what defines a skill is not so much how it was acquired or how difficult it is to perform but rather that the performance improves with practice. This section looks at different kinds of skill memories, exploring what factors determine how skill memories are acquired, recalled, improved, and sometimes lost.

Similarities Between Perceptual-Motor Skills and Cognitive Skills

As discussed in [Chapter 1](#), philosophers such as Aristotle and Descartes drew sharp distinctions between perceptual-motor skills and cognitive skills (thoughts). Psychologists similarly associate cognitive skills with the ability to reason and solve problems or to perform tasks (e.g., writing a textbook) that require sorting through large amounts of knowledge. Cognitive skills are commonly thought to depend more on intellectual prowess, whereas perceptual-motor skills depend more on physical dexterity, speed, and strength—the classic nerds-versus-jocks split. Solving math problems does clearly differ in many respects from playing football, yet recent evidence suggests that there are more similarities in how humans learn and remember both types of skills than was previously thought ([Rosenbaum et al., 2001](#)).

In addition to the similarities that exist between the cognitive and perceptual-motor categories of skills, there are similarities in the way researchers study them. Research on perceptual-motor skills typically focuses on much less complex skills than those needed to do backflips or play basketball. Skills studied in the laboratory might consist of pressing buttons quickly or tracking the position of a moving object ([Doyon, Penhune, & Ungerleider, 2003](#)). Likewise, psychologists studying cognitive skills often use tasks that participants can learn relatively quickly, such as simple puzzles or games. In both cases, researchers want to keep the task as simple as possible so they can control the relevant variables more precisely. This gives them a better chance of understanding how experience affects an individual's ability to perform a particular skill.

Real-world skills often involve a combination of perceptual-motor and cognitive components. For instance, mental focus, a cognitive skill related to cognitive control (a major topic of [Chapter](#)

9), is a vital component of gymnastics, especially at the Olympic level, where athletes must maintain their poise under tremendous pressure in order to be able to perform complex movements flawlessly. Psychologists classify skills such as gymnastics, which consist of performing a predefined sequence of movements, as [closed skills](#). Skills that require an individual to flexibly modify actions (or thoughts) in response to current circumstances are [open skills](#). Just as many activities require both perceptual-motor and cognitive skills, they also may demand a combination of open and closed skills ([Magill, 1993](#)). Passing a basketball involves particular movement patterns, but it also requires the players to adjust their movements flexibly in response to the length of the pass and the movements of other players.



George Gojkovich/Getty Images

What closed skills and open skills helped John Urschel become both a professional football player and an expert mathematician?

closed skill

A skill that involves performing predefined movements that, ideally, never vary.

open skill

A skill in which movements are made on the basis of predictions about changing demands of the environment.

Test Your Knowledge

Open and Closed Skills

Psychologists classify skills in many ways. One conventional scheme for classifying perceptual-motor skills is the extent to which skills are open or closed. Open skills involve movements that are modified based on predictions about environmental demands, and closed skills depend on performing predefined movements that, ideally, never vary. Try to label each of the following perceptual-motor skills as either an open skill or a closed skill, considering all the possible contexts in which the skills might be performed. ([Answers appear at the end of the chapter.](#))

1. A sea lion balancing a ball
2. A girl swimming
3. A couple kissing
4. A bear catching a fish
5. A fish catching insects
6. A boy playing a piano
7. A young woman throwing darts

Which Comes First: Cognitive or Perceptual-Motor Skill Memory?

The learning of new skills often begins with a set of instructions. Say you've just started college, and you hand your new roommate a bag of ramen to prepare for lunch. If your roommate has never seen ramen before, she might start scanning the bag for clues about what to do next. The makers of the ramen predicted that your roommate might not possess memories of how to cook ramen, so they provided written instructions about how to do it. A few weeks later, when your roommate decides to treat you to lunch, she may simply try to recall the ramen-making steps from memory. Because she will depend on her memories for events and facts to cook the lunch, you could say that her skill memories are her memories of the instructions or of a past episode. In other words, skill memories can be memories for events and facts!

Following a written recipe for how to prepare food requires the ability to read, which is a cognitive skill. Correctly following the instructions to make ramen also requires the ability to perform various perceptual-motor skills, such as opening the bag and boiling water. Both kinds of skill memories can contribute to learning new skills. Nevertheless, several lines of evidence

suggest that perceptual-motor skill memories provide the foundation for learning cognitive skills.

To begin with, humans learn perceptual-motor skills at younger ages than they learn cognitive skills. (The development of various memory abilities across the lifespan is discussed in detail in [Chapter 12](#).) Second, many cognitive skills, including reading and writing, are difficult or impossible to acquire without first learning basic perceptual-motor skills such as producing speech or drawing lines. Finally, many different species can learn to perform a wide range of perceptual-motor skills, as described in [Chapter 5](#), but only a few seem to be able to learn cognitive skills. In fact, many researchers and philosophers have argued that humans are the only organisms that can learn cognitive skills and that this ability is what makes humans special.

Can Nonhumans Have Cognitive Skill Memories?

René Descartes, who believed that many of our mental abilities are innate (see [Chapter 1](#)), proposed that the inborn ability to reason is what distinguishes humans from other animals. Descartes would probably have been willing to accept that dogs can store memories for how to perform perceptual-motor skills such as how to catch a Frisbee, but he would have considered it impossible for a dog or any other nonhuman animal to learn a cognitive skill. Following Descartes's lead, many early psychologists assumed that only humans can reason or perform complex cognitive tasks. Consequently, most of what is currently known about cognitive skill memories comes from studies of humans.

However, it is increasingly evident that humans are not the only animals that can learn cognitive skills after all. For instance, it was once thought that only humans use tools and that this particular problem-solving ability played a key role in the evolution of the human mind. But in the past two decades, psychologists and animal behavior researchers have described tool use in many animals, including elephants, apes, octopuses, birds, otters, and dolphins ([Beck, 1980](#); [Hart, 2001](#); [Hunt, Corballis, & Gray, 2001](#); [Krutzen et al., 2005](#); [Whiten et al., 1999](#)). In the lab, experimenters also have taught primates and other animals to use various human-made tools. There is even evidence that animals in the wild can teach themselves to use novel tools; for example, dolphins have learned to use sponges to facilitate foraging, as shown in [Figure 8.1](#) ([Krutzen et al., 2005](#)). The dolphins don't use sponges like the kind you can buy at a store, but they do collect natural sponges, which they have learned to use as helmets when they are capturing fish that hide among spiny sea urchins.



Yann hubert/Shutterstock

FIGURE 8.1 Dolphins' use of tools Some dolphins in Australia have taken to carrying around sponges when they are foraging. Researchers suspect that the dolphins use the sponges as tools to protect themselves against injuries from sea urchins and other spiny sea creatures as they probe the seafloor for food.

Researchers have also observed chimpanzees in the wild learn how to use stones to crack nuts ([Whiten & Boesch, 2001](#)). In some cases, chimpanzees will carry the stones long distances before actually using them. Tool use is an ability that typically involves both perceptual-motor and cognitive skills. Movement patterns required for using a tool improve with practice, as does the recognition that a particular tool (or strategy) can be useful in solving various problems.

Clearer evidence of cognitive skills in nonhumans comes from laboratory studies showing that various species can learn to perform cognitive tasks on command. In one study, mentioned briefly in [Chapter 7](#), dolphins learned to repeat whatever action they had performed most recently when instructed to do so by a trainer ([Mercado, Murray, Uyeyama, Pack, & Herman, 1998](#)). Most of the “tricks” that you may have seen dolphins performing on TV or in oceanariums are perceptual-motor skills. The trainer gives a signal, and in response the dolphin performs a particular movement sequence. When a dolphin is instructed to repeat an action,

however, the signal is not one that identifies a specific action; rather, it is a general signal that tells the animal to repeat whatever the prior action was. So almost any action a dolphin can perform could potentially be a correct response, depending on what the dolphin was doing before the instruction was given.

To successfully follow a “repeat” instruction, the dolphin must actively recall its recent actions. Recall or reenactment of past episodes is a cognitive skill: it can be improved through practice (as exemplified by the memory athletes described in [Chapter 7](#); [Brehmer, Li, Muller, von Oertzen, & Lindenberger, 2007](#)). Dolphins’ success at learning to respond accurately to a “repeat” command shows they can perform the cognitive skill of recalling past events.

Not all animals are equally capable of learning complex cognitive and perceptual-motor skills, however. By comparing different animals’ abilities to learn skills and by exploring which neural systems they use when forming and retrieving memories of different skills, scientists are gaining a clearer understanding of how skill memories work ([Mercado, 2008](#)).

Encoding New Memories

Now that you know how researchers classify different kinds of skills, let’s consider the question of what allows some individuals to excel at a particular skill. You will not be surprised to hear that practice is an important factor. We will examine how different kinds of practice affect performance and retention of skill memories, as well as why individuals who are great at one skill are not necessarily as good at other, similar skills.

More Repetition Does Not Guarantee Improvement

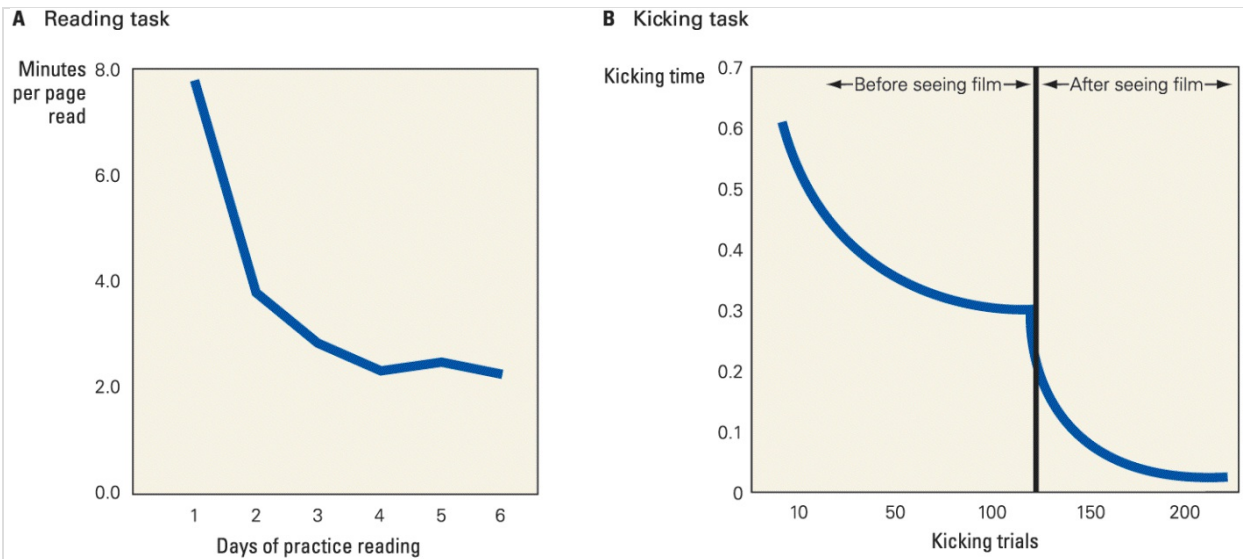
In the 2010 movie remake of *The Karate Kid*, a young boy asks a kung fu master to give him a crash course in martial arts. The master reluctantly agrees and begins by making the student repeatedly drop his coat on the floor, pick it up, put it on, take it off, and hang it up on a peg. This goes on for several days. The student does as he is told and later discovers that the movements he has been laboriously repeating are the kung fu movements that he needs to perform to defend himself. Because he has repeated these movements thousands of times, he is able to reproduce them rapidly and effortlessly. He (supposedly) has learned the skills of kung fu without even knowing it!

The portrayal of the relationship between practice and skill memories in this movie is similar to several early psychological theories of skill learning. The basic idea was that the more times you perform a skill, the faster or better you will be able to perform it in the future. Is this how practice works? To answer this question, Edward Thorndike repeatedly asked blindfolded individuals to draw a line exactly 3 inches long ([Thorndike, 1927](#)). Half of the participants were told when their line was close to the target length, and the other half were not given any feedback about their lines. Both groups drew the same number of lines during the experiment, but only the participants who received feedback improved in accuracy as the experiment progressed. This simple study suggests that repeatedly hanging up a coat is probably not a good way to learn kung fu moves. Feedback about performance, or **knowledge of results**, is critical to the effectiveness of practice ([Butki & Hoffman, 2003](#); [Ferrari, 1999](#); [Liu & Wrisberg, 1997](#); [Schmidt & Lee, 2005](#); [Turner & Martinek, 1999](#); [Weeks & Kordus, 1998](#)). Coaches, professors, and kung fu instructors all provide learners with knowledge of the results of practice (including studying), which can play a major role in how an individual's skills improve.

knowledge of results

Feedback about performance of a skill, critical to the effectiveness of practice.

Another general rule seems to be that the extent to which practice can improve performance decreases as the amount of practice increases ([Holding, 1981](#)). For example, **Figure 8.2A** shows that as participants practiced a reading task, the amount of time they required to read each page decreased ([Newell & Rosenbaum, 1981](#)). Early in training, participants showed large gains in reading speed, but after this initial improvement, each additional practice session led to minimal further improvement. This pattern is known as the **power law of practice**. It holds for a wide range of cognitive and perceptual-motor skills.



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

FIGURE 8.2 Effects of practice and feedback on skill performance (A) As training and practice at a reading task progressed, improvements in reading speed became smaller. (B) After improvement slows, new sources of feedback can lead to a new burst of rapid improvement. In this graph, after his initial rate of improvement in speed of kicking had begun to slow, a participant was shown a film of optimum kicking. The film helped him improve his own kicking speed.

(A) Data from [Singley & Anderson, 1989](#). (B) Data from [Hatze, 1976](#).

Description

The graph labeled A shows a learning curve for reading tasks. The horizontal axis plots days of practice reading and vertical axis plots minutes per page read. The readings on the horizontal axis range from 1 to 6 in increments of 1 and the readings on the vertical axis range from 0.0 to 8.0 in increments of 0.2. The curve starts at nearly 8 minutes per page read on Day 1, drops quickly to 4 minutes per page read on Day 2, 3 minutes per page read by Day 4, and 2 minutes per page read by Day 6.

Graph B shows the Kicking task. The horizontal-axis plots kicking trails and vertical-axis plots kicking time. The readings on the horizontal axis range from 0 to 200 in varying increments and the readings on the vertical axis range from 0 to 0.7 in increments of 0.1. A vertical line at point 125 on the horizontal axis, divides the graph into two planes- before seeing the film and after seeing the film. The curve starts at 0.6 for the first trial and descends after that meeting the vertical line. Thereon, the curve descends even more steeply and finally plateaus at around 175 trials.

power law of practice

A law stating that the degree to which each new practice session improves performance diminishes after a certain point, such that greater numbers of sessions are needed to further improve the skill; learning occurs quickly at first and then slows down.

When you first learned to use a computer keyboard, you had to search for keys and were probably pretty slow. After your first year of using a keyboard, you likely doubled or tripled the number of words you could type per minute. If your typing speed doubled after every year of

practice, you would be typing incredibly fast by now! But the power law of practice predicts that this will not happen. According to the power law, each additional year of practice after the first year produces smaller increases in typing speed; learning occurs quickly at the start and then slows down.

The power law of practice provides a useful description of how practice generally affects performance. It is possible to override this law, however, and enhance the effects of continued practice. For example, in one experiment, researchers asked a participant to kick a target as rapidly as possible. With visual feedback about his kicking speed, the participant was able to gradually kick faster, but eventually his improvement slowed and stopped, exactly as the power law of practice would predict ([Hatze, 1976](#)). At that point, the researchers showed him a film comparing his movements with movements known to minimize kicking time. After seeing the film, the man again improved his kicking speed considerably ([Figure 8.2B](#)). This is an example of *observational learning*, a topic we discuss in detail in [Chapter 11](#). The participant observing the film formed memories of the observed kicking technique that he later used to improve his own performance.

The kinds of feedback provided during practice can strongly affect when and if performance improves. Experiments show that frequent feedback in simple perceptual-motor tasks leads to good performance in the short term but mediocre performance in the long term, whereas infrequent feedback leads to mediocre performance in the short term but better performance in the long term ([Schmidt & Wulf, 1997](#); [Schmidt, Young, Swinnen, & Shapiro, 1989](#)). Instructors and coaches have sometimes discovered through trial and error what types of feedback work best. For instance, dance instructors found that the visual feedback provided by mirrors enhances the effects of practicing dance movements, and most dance studios now have mirrors on the walls.

Can you think of any similar advances that college professors have made in the past century in providing feedback to improve students' cognitive skills? One example might be online tutorials that provide immediate feedback about the accuracy of students' responses to intermittent quizzes; some research suggests that these can produce faster learning and greater achievement levels than classroom instruction ([Anderson, Corbett, Koedinger, & Pelletier, 1995](#)).



Taiyou Nomachi/DigitalVision/Getty Images

What are some ways that the dancing of the teacher and other students may provide feedback that can enhance the benefits of practice?

Timing and Sequencing of Practice Matters

To summarize, feedback is critical to the acquisition of skill memories because it affects how individuals perform during practice; certain forms of information that precede practice, such as instructional videos, can have similar effects. But skill memories do not depend only on the way skills are taught and practiced. They also depend on how practice time is apportioned.

Concentrated, continuous practice, or **massed practice**, generally produces better performance in the short term, but **spaced practice**, which is spread out over several sessions, often leads to better retention in the long run ([Arthur et al., 2010](#)).

massed practice

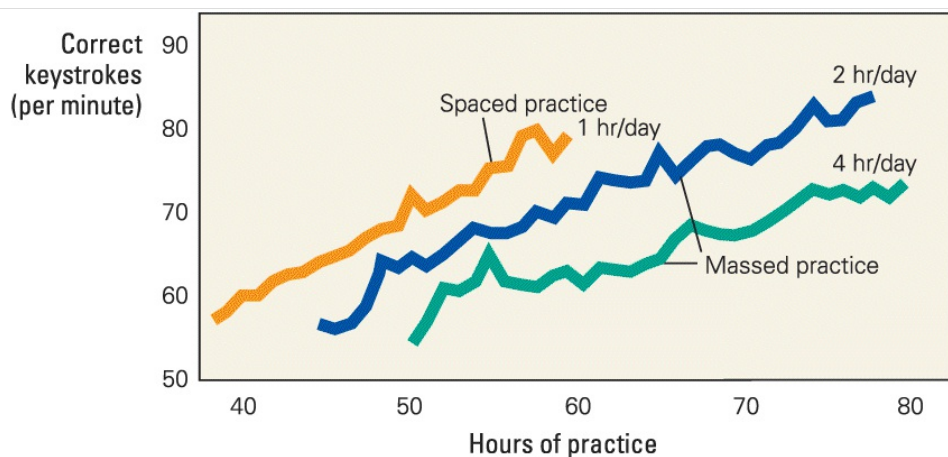
Concentrated, continuous practice of a skill.

spaced practice

Practice of a skill that is spread out over several sessions.

Consider the following classic experiment. Three groups of post office workers were trained to use a keyboard to control a letter-sorting machine. One group practiced 1 hour a day for 60 days.

The other two groups practiced either 2 hours a day for 40 days or 4 hours a day for 20 days ([Baddeley & Longman, 1978](#)). Contrary to what you might guess, the group that practiced for only 1 hour a day (spaced practice) required fewer total hours of training than any other group to become proficient at using the keyboard ([Figure 8.3](#)). The downside was that this group had to be trained over a longer period—2 months instead of 1. Interestingly, when participants were surveyed about their satisfaction with their training schedule, those who trained for 1 hour a day were the least satisfied, whereas those who trained for 4 hours a day were the most satisfied, suggesting that satisfaction with a training program (or course) may not be a very good measure of the learning achieved in that program. Although researchers have conducted many studies to determine what kinds of practice schedules lead to optimal learning and performance, there is still no general consensus about how to identify the best possible schedule for any given individual attempting to learn any particular skill.



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FIGURE 8.3 Benefits of spaced practice versus massed practice The performance of post office workers using a keyboard to control a letter-sorting machine improved at different rates, depending on their training schedules. Workers who practiced for 1 hour a day for 60 days (spaced practice) improved their performance more per hour of practice than did workers who practiced for 2 or 4 hours a day for 40 and 20 days, respectively. Although the group with spaced practice learned the task in fewer total hours, the training took longer (2 months).

Data from [Baddeley & Longman, 1978](#).

Description

The horizontal axis plots hours of practice ranging from 40 to 80 hours in increments of 10. The vertical axis plots correct keystrokes (per minute), ranging from 50 to 90 in increments of 10. The graph has three curves representing massed practice (2 hours per day), massed practice (4 hours per day), and spaced practice (1 hour per day).

The spaced practice curve starts at 58.35 correct keystrokes per minute and gradually increases to 80 correct

keystrokes per minute, at about 60 hours of practice.

The 2 hours per day massed practice curve starts at 55 correct keystrokes per minute and 45 hours of practice. It gradually increases to 82 correct keystrokes with 76 hours of practice.

The 4 hours per day massed practice curve starts at 55 correct keystrokes per minute and 50 hours of practice. It gradually increases to 70 correct keystrokes with 80 hours of practice.

All the data is approximate.

Researchers also observe differences in the outcomes after equal amounts of practice when they compare different *kinds* of practice (as opposed to different *schedules* of practice). In this case, the comparison is between **constant practice**, which involves a very limited set of materials and skills, and **variable practice**, which involves more varied materials and skills. Constant practice consists of repeatedly practicing the same skill—for example, repeatedly attempting to throw a dart at the bull’s-eye of a dartboard under fixed lighting conditions or attempting to master a single trick shot in pool. Variable practice consists of practicing a skill in a wider variety of conditions, such as attempting to hit each number on a dartboard under various levels of lighting or trying to improve one’s performance at interviews by applying for a diverse range of jobs.

constant practice

Practice involving a constrained set of materials and skills.

variable practice

Practice involving the performance of skills in a wide variety of contexts.

Several studies have shown that variable practice leads to better performance in later tests (and can also enhance generalization, as [Chapter 6](#) describes). In one such experiment, individuals tracked targets that were moving along various paths. People who used variable practice to learn this task performed better, both in training sessions and in later tests, than did individuals who trained with constant practice ([Wulf & Schmidt, 1997](#)). Variable practice is not always more effective than constant practice, however ([van Rossum, 1990](#)); researchers have not yet discovered how to reliably predict when variable practice will lead to better learning and performance.

In fact, variable practice can sometimes lead to slower progress than constant practice. Even in these cases, however, later performance usually is superior following variable practice ([Schmidt & Bjork, 1992](#)). Just as the benefits of spaced training were not obvious to participants learning

keyboard skills in the study depicted in [Figure 8.3](#), the benefits of variable training are not always apparent during variable practice. One type of variable practice that has been shown to be particularly effective involves gradually increasing the difficulty of trials during training ([Kagerer, Contreras-Vidal, & Stelmach, 1997](#); [Sawers, Kelly, & Hahn, 2013](#)). During gradual training, performance is initially easy and then is made incrementally more difficult as training progresses. Intuitively, you might think that spending several hours practicing a difficult task would lead to more improvement on that task than spending most of those hours practicing easier versions of the task followed by only a few hours practicing the actual task you need to perform. For many tasks, however, the opposite is true. Gradual training often leads to better overall performance, despite the fact that the practice trials require less effort overall.

Musicians take advantage of this effect by initially practicing musical pieces at slower rates than they will ultimately be required to play them, as well as by practicing various musical scales. In short, the timing, variability, and ordering of training sessions can be as important as the quality of feedback and amount of practice. Surprisingly, expending less effort (gradual training) spread out over longer periods (spaced practice) can enhance skill acquisition more than grueling, nonstop practice can.

Skill Memories Are Often Formed Unconsciously

When you acquire a skill, it is usually because you have made a conscious effort to learn the skill over time. It is possible to “accidentally” learn to perform certain skills, however, without ever being aware of what you have learned. You probably would not be able to master kung fu without knowing that you are learning the moves, but you might be able to learn to be better at kissing or conversing without being aware that you are doing anything differently. Learning that happens incidentally, without awareness of what has been learned, is called **implicit learning** ([Seger, 1994](#)). Implicit learning produces implicit memories, which are defined in [Chapter 7](#) (and mentioned above with reference to H.M.) as memories that are acquired without conscious awareness. Given this lack of conscious awareness, you would be hard pressed to estimate how many skills you have acquired in this way. For all you know, you are implicitly learning right now!

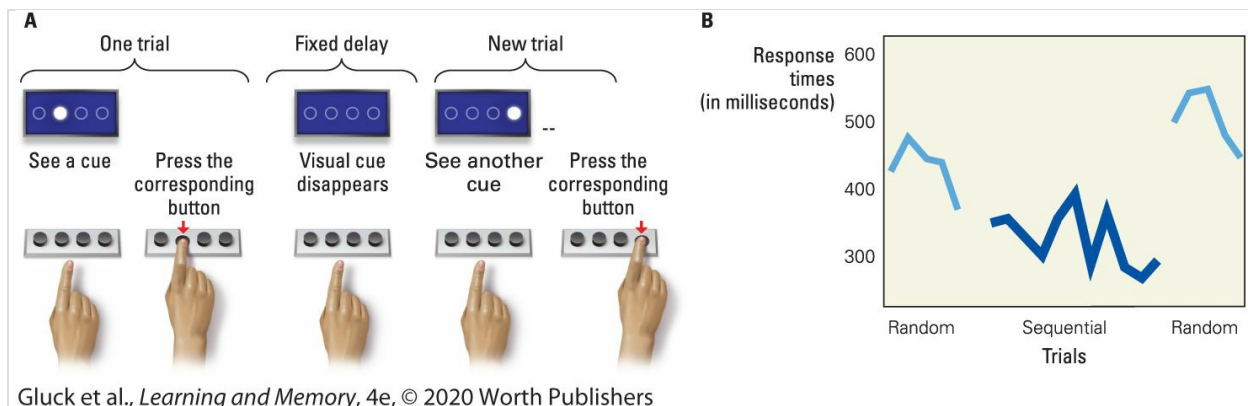
implicit learning

Learning that occurs without the learner’s awareness of improvements in performance or, in the case of people with amnesia, awareness that practice has occurred.

Studies of individuals with anterograde amnesia (like H.M.) provided early evidence of implicit skill learning ([Knowlton et al., 1996](#); [Pohl, McDowd, Filion, Richards, & Stiers, 2001](#); [Willingham, 1999](#); [Wulf & Schmidt, 1997](#)). [Chapter 7](#) describes the difficulty that individuals with anterograde amnesia (including H.M.) have with learning and remembering events and facts. However, such individuals can acquire new skills relatively normally, showing improvement from one session to the next even while claiming that they have never practiced or observed the skill in the past ([Cohen, Poldrack, & Eichenbaum, 1997](#); [Seger, 1994](#); [Sun, Slusarz, & Terry, 2005](#)). Individuals with anterograde amnesia make an effort to learn the skill during each session but always think they are trying it for the first time. The fact that their performance improves with each session demonstrates that they are forming skill memories even though they can't verbally describe prior practice sessions. H.M. was able to learn new perceptual-motor skills, but he did not know that he had learned them ([Corkin, 2002](#); [Gabrieli, Corkin, Mickel, & Growdon, 1993](#); [Tranel, Damasio, Damasio, & Brandt, 1994](#)).

One need not be amnesic, however, to learn skills implicitly. You may perform some task, such as washing windows, and incidentally learn an underlying skill that facilitates performance of that task. For instance, maybe you learn that circular rubbing movements shine the window brighter and faster than random rubbing—yet you may or may not realize that you have discovered a faster, better approach.

To study implicit skill learning in people without brain damage, psychologists commonly use the **serial reaction time task**, in which participants learn to press one of four keys as soon as a visual cue (a light) indicates which key to press ([Figure 8.4A](#)). The computer presents the visual cues in long sequences that are either unpredictably ordered (called the *random condition*) or ordered in a fixed sequence of about 12 cues (called the *sequential*, or *implicit learning*, *condition*). For example, if we designate the four keys from right to left as A through D, then the fixed sequence might be ABADBCDACBDC. Participants eventually begin to get a feel for the repeating sequential patterns and anticipate which key to press next, as reflected by faster reaction times for implicitly learned sequences relative to random sequences ([Figure 8.4B](#)). When researchers interview participants after training, however, they typically show no awareness that any of the sequences were repeating patterns ([Exner, Koschack, & Irle, 2002](#)). In other words, they have learned the sequences without knowing that they learned them, making their learning implicit.



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FIGURE 8.4 Serial reaction time task in the study of implicit learning (A) In a serial reaction time task, participants learn to press keys as rapidly as possible in response to visual cues provided on a computer screen. (B) Participants' reaction times are slower when the cues are presented in random order than when they are presented in a fixed sequence. Quicker reaction times for the sequential cues indicate that the participants implicitly learned to anticipate which key they needed to press next, even though their verbal reports reveal no awareness that there was a fixed sequence.

Research from [Robertson, 2007](#).

Description

The illustration has three parts labeled as one trial, fixed delay, and a new trial.

One-trial: A display with four slots and a panel with four buttons are shown. The display flashes a cue in position 2. A finger is placed near the second button. In the next step, the second button corresponding to the cue is pressed by the finger.

Fixed delay: The visual cue disappears. The finger is positioned near the second button.

New trial: The cue is flashed in position 4. The finger presses the fourth button, corresponding to the cue.

The graph plots response time in milliseconds on the vertical axis against trials on the horizontal axis. The labels on the horizontal axis read, random, sequential, and random. The readings on the vertical axis range from 300 to 600 in increments of 100. The information in the graph is as follows.

Random trials: The response time starts from 410 milliseconds, ascends to 490 milliseconds, and drops to 380 milliseconds.

Sequential trials: The response time starts from 350 milliseconds, slightly increases to 370 milliseconds, after which the curve has sharp peaks and falls finally ending at 300 milliseconds.

Random: The response time starts from 500 milliseconds, rises to 520 milliseconds, after which it descends to 480 milliseconds.

serial reaction time task

An experimental task that requires individuals to press keys in specific sequences on the basis of cues provided by a computer; used to study implicit learning.

As mentioned earlier in this chapter and in [Chapter 7](#), people often have difficulty verbalizing what they have learned after mastering a perceptual-motor skill. This might seem to suggest that perceptual-motor skills are more likely than cognitive skills to be learned implicitly. But people can also acquire many components of cognitive skills through implicit learning. No one becomes a chess grand master simply by reading the rules of chess and listening to other players explaining why they made particular moves. Mathematical whizzes do not become experts by simply hearing about mathematical axioms and proofs ([Lewis, 1981](#)). Development of both these skills requires extensive practice, during which implicit learning undoubtedly occurs. In the case of cognitive skill acquisition, it is difficult to assess which abilities are improving unconsciously because the changes in thinking produced by practice are not easy to observe. Moreover, the learner would, by definition, be unaware of these changes and therefore unable to report them. Consequently, there is currently no way to assess whether implicit learning is more likely to occur during the learning of perceptual-motor or cognitive skills.

The distinction between implicit and explicit learning—one being unconscious, the other requiring conscious awareness—suggests that they may involve two quite different modes of forming skill memories. This distinction may not be as sharp as it seems, however ([Robertson, 2007](#); [Song, 2009](#)). For instance, you might become aware that you are improving at a skill before realizing what it is that you are doing differently, or you might be aware that your attempts at improving a skill (say, the skill of getting a second date) are not working without recognizing what you are doing wrong (monopolizing the conversation on the first date). In this case, implicit and explicit learning would correspond to points along a continuum of awareness rather than to two different kinds of learning.

There is also evidence suggesting that conscious awareness may be a prerequisite for achieving certain levels of expertise. For example, elite athletes often endure grueling practice sessions not because they enjoy such activities or because they are more talented and therefore have greater endurance than other athletes but because they are more aware of the necessity of such practice regimens for achieving the highest levels of performance ([Yarrow, Brown, & Krakauer, 2009](#)). So, although you may be able to learn skills without making a conscious effort to do so, you are unlikely to accidentally become a professional athlete, rock star, or kung fu master.

Becoming an Expert

When you practice a skill, you typically do so because you want your performance to become more controlled and effortless. If you are learning to juggle, you might ultimately be able to show off your mastery by juggling while casually talking to a friend. For an expert juggler, the skill has become almost automatic. Perceptual-motor skills that an organism can perform with minimal attention are called **motor programs** or, sometimes, *habits* ([Graybiel, 2008](#); [James, 1890](#)).



Brooke Slezak/The Image Bank/Getty Images

What stage of skill acquisition has this juggler achieved?

motor program

A sequence of movements that an organism can perform automatically (with minimal attention).

Cognitive skills, with extended practice, can also become motor programs. Paul Fitts, a psychologist whose studies focused on human movement, proposed that skill learning usually progresses through three stages ([Table 8.2](#)), including an initial period, called the [cognitive stage](#), when an individual must actively think to encode and perform a skill ([Fitts, 1964](#)). When your roommate makes ramen by following written instructions, she is in the cognitive stage of skill acquisition. If you are learning to juggle for the first time, then you will initially have to find out what the steps are and keep them constantly in mind. This, too, corresponds to the cognitive stage of skill learning.

cognitive stage

The first stage in Fitts's model of skill learning, when an individual must actively think to encode and perform a skill.

TABLE 8.2 Fitts's three-stage model of skill learning

Stage	Characteristics	Example
1. Cognitive stage	Performance is based on rules that can be verbalized.	Using written instructions to set up a tent
2. Associative stage	Actions become stereotyped.	Setting up a tent in a fixed sequence, without instructions
3. Autonomous stage	Movements seem automatic.	Setting up a tent while carrying on a discussion about politics

During the next stage, called the [associative stage](#), learners begin to use stereotyped actions when performing the skill and rely less on verbal memories of specific steps. The first few times you play a video game, for example, you may need to keep reminding yourself about the combinations of controller button presses that are necessary to produce certain actions. Eventually, however, you no longer need to think about these combinations. When you decide that you want a particular action to occur on the screen, your hands do what is necessary to make that action happen.

associative stage

The second stage in Fitts's model of skill learning, when learners begin to use stereotyped actions in performing a skill and rely

less on actively recalled memories of rules.

To reach high levels of skill performance, however, your movement patterns must become quick and effortless. Fitts described this third level of skill learning as the [autonomous stage](#)—the stage at which the skill or subcomponents of the skill have become motor programs. At this stage, thinking too much about what you are doing when you perform a skill can actually impair performance—a phenomenon related to “choking” under pressure. The three stages proposed by Fitts continue to be included in more recent models of skill acquisition ([Ackerman, 2007](#)).

autonomous stage

The third stage in Fitts’s model of skill learning, when a skill or subcomponents of the skill become motor programs.

Some psychologists argue that practice alone determines who will become an expert, suggesting that at least 10,000 hours of deliberate, quality practice is required ([Ericsson, Krampe, & Tesch-Romer, 1993](#); [Ericsson & Lehman, 1996](#)). Individuals who master complex skills (e.g., playing chess or a musical instrument) at an early age, however, are more likely to reach the highest levels of performance ([Howard, 2009](#)).

Researchers studying expertise have often focused on the abilities of athletes, chess masters, or other professional game players—for several reasons. First, people who learn to play games outside a research lab serve as good examples of “real-world” skill learning. Second, it is relatively easy to find people with widely varying levels of expertise in sports or games such as chess, and relative ability can be quantified based on performance in competitions. Finally, sports and games require a variety of perceptual-motor and cognitive skills, making them useful for investigating many different psychological phenomena.



Patrick Hickey /www.CartoonStock.com

Simon had mastered clicking his mouse while he slept, but he would soon learn this was useless without the correct posture.

Description

A man is leaning backward and sleeping during office hours. His mouth is partly open, and he has his left hands on the mouse at his desk. Another man is silently standing behind him. One more man is working in a cubicle nearby.

Researchers studying expert chess players found that experts scan the game board (a visual-motor skill) differently than less experienced players do ([Charness, Reingold, Pomplun, & Stampe, 2001](#)). When chess masters look at chess pieces, their eyes move rapidly to focus on a small number of locations on the board, whereas amateur chess players typically scan larger numbers of locations and do so more slowly. When experts stop moving their eyes, they are more likely than nonexperts to focus on empty squares or on strategically relevant chess pieces. Differences in visual scanning are also seen in expert athletes. Amateur soccer players tend to watch the ball and the player who is passing it, whereas expert players focus more on the movements of players who do not have the ball ([Williams, Davids, Burwitz, & Williams, 1992](#)). This finding suggests that both perceptual-motor and cognitive skills may contribute to the superior abilities of experts.

Talent Blossoms with Practice

Different individuals start with different capabilities, and the extent to which practice can

improve their performance levels also varies from one person to the next. People who seem to master a skill with little effort (the way Mozart mastered anything related to music) are often described as having a **talent**, or “gift,” for that skill. The people who start off performing a skill well are often those who end up becoming experts ([Howard, 2009](#)), but someone who initially has little ability to perform a skill may, with practice, become better at that skill than someone who seemed destined to become a star.

talent

A person’s genetically endowed ability to perform a skill better than most.

What role does talent play in achieving expertise in cognitive or perceptual-motor skills? Even child prodigies are not born with the ability to perform the skills that make them famous. Like everyone else, they learn to perform these skills. Mozart’s father, a professional musician, trained Mozart extensively from a young age. So it is difficult to determine to what extent Mozart’s musical abilities were a result of his musical talents and to what extent they were a result of his father’s teaching abilities and persistence. Most modern research equates talent with genetic predispositions that positively affect an individual’s acquired abilities.

Psychologists have attempted to gauge the role of genetics in skill learning and performance by conducting studies with twins—some identical (who share 100% of their genes) and some fraternal (who, like other siblings, share only about 50% of their genes). Some of these studies examine pairs of twins who were raised in different homes to gauge the effects of environment. In one large study of twins reared apart, researchers at the University of Minnesota trained participants to perform a skill in which they had to keep the end of a pointed stick, called a stylus, above a target drawn on the edge of a rotating disk, as shown in [Figure 8.5A](#) ([Fox, Hershberger, & Bouchard, 1996](#)). Researchers frequently use this task, known as the **rotary pursuit task**, in laboratory studies of perceptual-motor skill learning. The task requires precise hand–eye coordination. When individuals first attempt the rotary pursuit task, they generally show some ability to keep the stylus over the target but must adjust the speed and trajectory of their arm movements fairly often to correct for errors (e.g., lagging behind the target). With additional practice, most individuals rapidly improve their accuracy, increasing the amount of time they can keep the stylus tip over the target ([Figure 8.5B](#)).

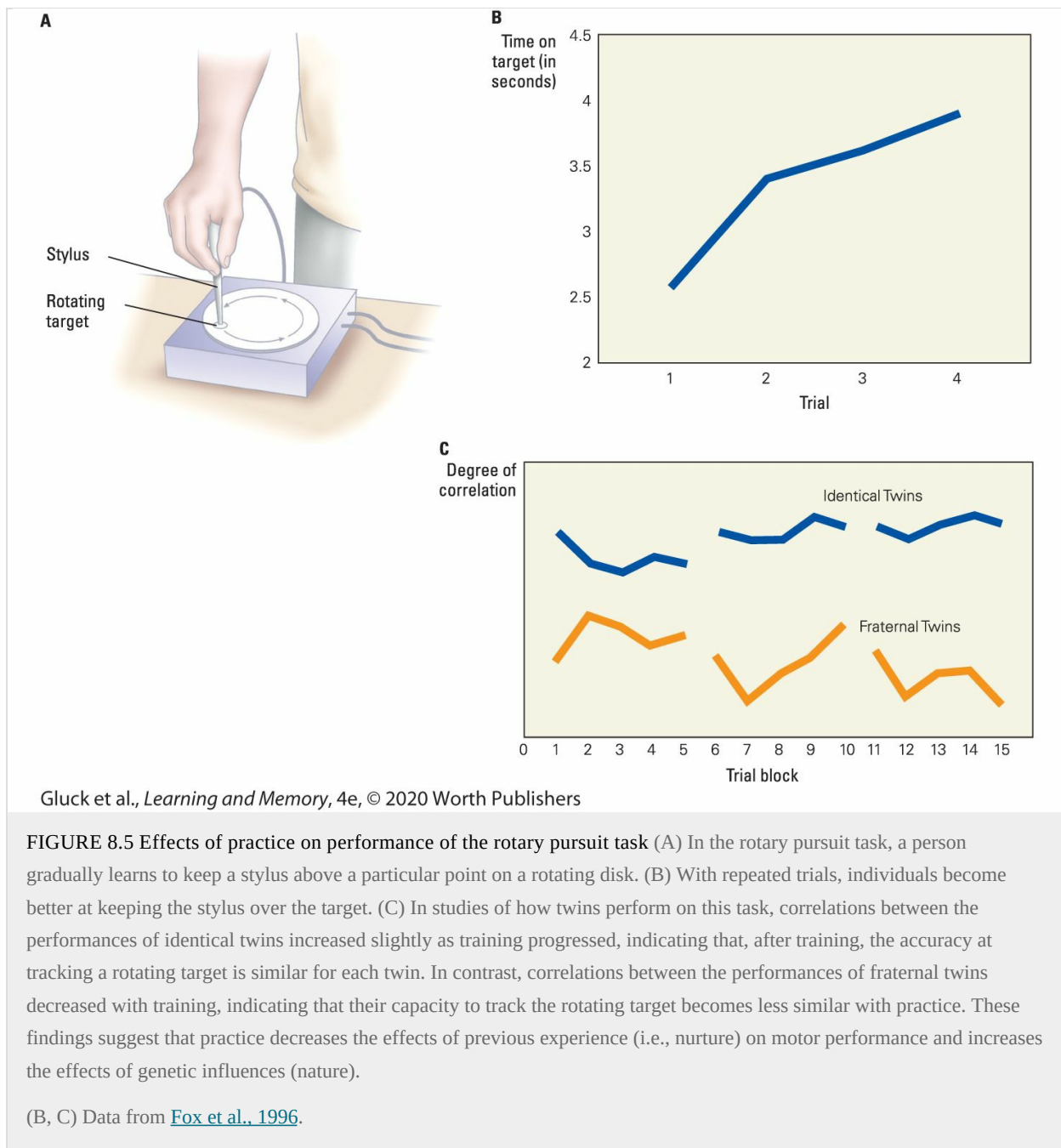


FIGURE 8.5 Effects of practice on performance of the rotary pursuit task (A) In the rotary pursuit task, a person gradually learns to keep a stylus above a particular point on a rotating disk. (B) With repeated trials, individuals become better at keeping the stylus over the target. (C) In studies of how twins perform on this task, correlations between the performances of identical twins increased slightly as training progressed, indicating that, after training, the accuracy at tracking a rotating target is similar for each twin. In contrast, correlations between the performances of fraternal twins decreased with training, indicating that their capacity to track the rotating target becomes less similar with practice. These findings suggest that practice decreases the effects of previous experience (i.e., nurture) on motor performance and increases the effects of genetic influences (nature).

(B, C) Data from [Fox et al., 1996](#).

Description

The illustration in section A shows a close-up view of a person's hand holding a stylus and pressing it against a cylindrical rotating target.

The graph in section B plots time on target in seconds on the vertical axis against trials on the horizontal axis. The readings on the vertical axis range from 2 to 4.5 in increments of 0.5 and the trials are numbered from 1 through 4. A line starts at point (1, 2.5), ascends to (2, 3.5), passes through (3, 3.7), and ends at (4, 4.0).

The graph in section C plots the degree of correlation on the vertical axis against the trial block on the horizontal axis.

The trial blocks are numbered from 0 to 15 in increments of 1. The graph shows two sets of curves representing identical twins and fraternal twins. The curves representing identical twins are above the curves representing fraternal twins. Identical twins have a similar pattern in keeping the stylus on the target. The fraternal twins show a dissimilar pattern in keeping the stylus on the target. Identical twins show a higher degree of correlation than fraternal twins.

rotary pursuit task

An experimental task that requires individuals to keep the end of a pointed stick (stylus) above a fixed point on a rotating disk; used to study perceptual-motor skill learning.

The researchers found that when they trained twins to perform the rotary pursuit task, identical twins' abilities to keep the stylus on the target became more similar as training progressed, whereas fraternal twins' abilities became more dissimilar. That is, during training, the performance of one twin became more correlated with the performance of the second twin when the two twins shared 100% of their genes ([Figure 8.5C](#)). Put another way, if you were to view videos of the participants' hands after training as they attempted to keep the stylus above the rotating target, you would judge the movements of identical twins' hands to be the most similar. Their movements might remind you of synchronized swimming. In the case of fraternal twins, however, you would probably judge their movements after training to be dissimilar. For example, one twin might keep the stylus over the target continuously, while the other twin might have to increase her speed a bit every few seconds to catch up with the target.

One interpretation of what is happening in identical twins as they learn the rotary pursuit task is that practice decreases the effects of prior experiences on the accuracy of their tracking movements and increases the effects of genetic influences. In other words, the more people practice a skill, the more individual differences in their performance are due to genetic differences. Identical twins have the same genes, so when practice increases the contributions of genes to their perceptual-motor skills, their performances become more similar. Perhaps future genetic analyses will discover biological correlates of specific talents, thus permitting identification of individuals who have inherited propensities to perform certain skills exceptionally well. (See "[Learning and Memory in Everyday Life](#)" for a brief consideration of the interplay of genes and environment in determining what men and women find easy to learn.)

LEARNING AND MEMORY IN EVERYDAY LIFE

Are Some Cognitive Skills Easier for Men Than for Women?

In 2005, the president of Harvard University, Larry Summers, provoked a national uproar when he suggested that one

reason there are more men than women in top-tier science and engineering positions is because of “different availability of aptitude at the high end.” Essentially, he was suggesting that, among the most scientifically or technologically talented people that are born, there are more men than women. If this claim had been made in relation to athletic skills, then it probably would not have made a ripple, because one need only consult the record books (or rosters of professional sports leagues) to see that the fastest sprints, highest jumps, and longest shot puts were all performed by men.

This does not mean that men are able to learn to perform all (or even most) perceptual-motor skills at levels beyond what is possible for women, but it certainly supports the idea that there are some biological qualities (e.g., strength, height) that give men an edge when it comes to reaching the upper echelons of athletic performance. Might something similar be true for cognitive skill learning by males?

If one examines the historical record of scientific and technological achievements, it is clear that men have received more accolades and high-level positions than women. Unlike the findings in modern sports, however, this pattern could easily reflect sociocultural constraints rather than intrinsic differences in capacities. Stronger evidence of sex differences in cognitive performance comes from standardized high school achievement exams such as Advanced Placement (AP) tests. In these tests, substantially more men receive high scores in calculus, chemistry, and physics, despite the fact that more women take the tests ([Ackerman, 2006](#)). But these data do not show that men are better able to learn about scientific topics; they show merely that men currently are acquiring more cognitive skills related to retrieving and applying scientific knowledge during their early education. The resulting greater facility with scientific knowledge would make the learning of related cognitive skills in college courses easier; in this sense, then, scientific and technological skills would be easier for the most capable male undergraduates to learn than they would be for the most capable females.

However, there is currently no way to assess whether these differences in performance reflect differences in the cognitive talents of men versus women or whether they reflect differences in the interests, motivation, instruction, or practice of men versus women when learning about science and technology. Until more is known about how neural mechanisms constrain skill learning capacity, it may be impossible to assess which features of male or female brains might enhance (or depress) learning of different cognitive skills.

Retrieving Existing Memories

Skill memories are often highly restricted in terms of how they can be used ([Brady, 2008](#); [Goodwin, Eckerson, & Voll, 2001](#); [Goodwin & Meeuwse, 1995](#); [Ma, Trombly, & Robinson-Podolski, 1999](#)). You may have mastered the culinary skills needed to make great ramen, but this will not make you a great sushi chef. In some cases, skill memories are so specific that the introduction of additional informative cues can disrupt retrieval. For example, after individuals were trained to touch a target without receiving visual feedback about their arm movements, their performance was *worse* when researchers allowed them to see their arm moving as they carried out the task ([Proteau, Marteniuk, & Levesque, 1992](#)). Most people normally use visual feedback when learning to aim at a target, so it is surprising that providing such feedback can actually interfere with performance of a skill.

In other cases, skills seem to transfer to novel situations, a phenomenon called **transfer of training**. For instance, you learned to write with your right or left hand, and you may even have practiced with each hand, but have you ever written with your mouth or feet? If you try, you will discover that you can write semi-legible text using these and other body parts. You are able to transfer what you have learned about writing with one hand to other body parts, despite large differences in the specific movements you must perform to do so. In sports, teams spend much of their time practicing in scrimmages, with the hope that these experiences will transfer positively to similar situations in real games, even if the exact identities and positions of players are different in the real game than in field practice. If skills learned during practice did not transfer to real games, it is unlikely that so many coaches in so many different sports would train their teams in this way.

transfer of training

The transfer of skill memories to novel situations.

The restricted applicability of some learned skills to specific situations is known as **transfer specificity**. This phenomenon led Thorndike to propose that the transfer of learned abilities to novel situations depends on the number of elements in the new situation that are identical to those in the situation in which the skills were encoded ([Thorndike & Woodworth, 1901](#)). Thorndike's proposal, called the **identical elements theory**, provides one possible account of why transfer specificity occurs. It predicts that a tennis player who trained on hard courts might suffer a bit the first time she attempted to play on a clay court and would do progressively worse as the game was changed from tennis to badminton or table tennis (though she still might outperform an opponent who had never learned any racquet sport). Conceptually, transfer specificity is closely related to transfer-appropriate processing, which is described in [Chapter 7](#). The main conceptual difference is whether the memories being recalled are memories of skills or memories of facts and events.



Janet Kimber/The Image Bank/Getty Images

Will practicing the Guitar Hero video game help this man improve his skill at playing a real guitar?

transfer specificity

The restricted applicability of learned skills to specific situations.

identical elements theory

Thorndike's proposal that learned abilities transfer to novel situations to an extent that depends on the number of elements in the new situation that are identical to those in the situation in which the skills were encoded.

When performing a skill that you have learned in the past, you are generalizing from a past experience to the present. (Generalization is discussed in detail in [Chapter 6](#).) Since no two life experiences are ever identical, performing a skill always involves transfer of training. For example, each time you open a door, you are making use of skill memories you have acquired by opening doors in the past. Opening a door can require combinations of complex actions: grasping, inserting, and turning a key; grasping, pushing, pulling, or sliding a handle; and knob turning, latch unhooking, and so on. At this point in your life, you have probably encountered so many different doors that you are able to transfer those skills to open almost any door you encounter.

Acquiring the ability to learn novel tasks rapidly based on frequent experiences with similar tasks is called **learning set formation** ([Harlow, 1949](#)), or learning-to-learn. Learning set formation occurs in infants as they learn basic perceptual-motor skills ([Adolph & Joh, 2009](#)), as well as in adults as they learn more complex motor skills ([Braun, Mehring, & Wolpert, 2010](#); [Ranganathan, Wieser, Mosier, Mussa-Ivaldi, & Scheidt, 2014](#)). Learning set formation can also play an important role in the development and application of cognitive skills. For instance, after years of practice, professional actors acquire the ability to quickly adopt the personas of fictional characters and to memorize detailed scripts and action sequences in a relatively short period. To do so, they implicitly use many of the strategies that are known to benefit memory encoding, such as deep processing during encoding (discussed in [Chapter 7](#)) and chunking (discussed in [Chapter 9](#)). This suggests that at least one of the skills that actors acquire and transfer flexibly throughout their careers is the ability to rapidly encode and recall dialogue ([Noice & Noice, 2006](#)).

learning set formation

Acquisition of the ability to learn novel tasks rapidly based on frequent experiences with similar tasks.

TEST YOUR KNOWLEDGE

Tasks That Test Skill Memory

Several tasks are commonly used to investigate different phenomena related to skill memory, including the serial reaction time task and the rotary pursuit task. Identify which aspects of skill memories each of these tasks has been useful for testing. ([Answers appear at the end of the chapter.](#))

When Memory Fails

Like the strength of memories for facts and events, the persistence of a skill—that is, how well the skill, after being acquired, is performed on a later occasion—depends on the complexity of the skill, how well the skill memory was encoded in the first place, how often the skill has subsequently been performed, and the conditions in which recall is attempted ([Arthur, Bennett, Stanush, & McNelly, 1998](#)). The common belief that you never forget how to ride a bicycle once you learn to do it is not accurate. Although skill memories can last a lifetime, they do deteriorate with non-use. Generally, retention of perceptual-motor skills is better than retention of cognitive skills, but unless you actively maintain your bike-riding skills, the skill memories you created when you first learned to ride will gradually deteriorate.

Researchers have studied the forgetting of events and facts much more than they have studied the forgetting of skills. Perhaps this is because if someone loses the ability to do something, it is hard to judge whether she has forgotten how to do it, has forgotten that she knows how to do it, or has lost the physical control or strength necessary to perform what she recalls. Loss of motor control does not imply that a skill memory is forgotten. (In other words, a pianist experiencing temporary paralysis may not be able to play an arpeggio, but she has lost only the ability to employ the skill and not the skill itself.) Yet to the outside observer, it may be impossible to distinguish whether someone knows how to perform a skill but has impaired movement abilities or whether the person has forgotten how to perform the skill.

Psychologists call loss of a skill through non-use **skill decay**. Most research to date indicates that skill decay follows patterns similar to those seen in the forgetting of memories for events and facts. Motor deficits and injuries (including brain damage) often promote skill decay because they lead to non-use of learned skills.

skill decay

Loss of a skill due to non-use.

In some ways, forgetting a skill is like learning it in reverse. Not performing the skill is almost the opposite of practice: if you don't use it, you lose it ([Ford, Baldwin, & Prasad, 2018](#)). Most forgetting occurs soon after the last performance of the skill; as time goes by, less and less forgetting occurs. Thus, forgetting curves are similar to learning curves. Forgetting occurs

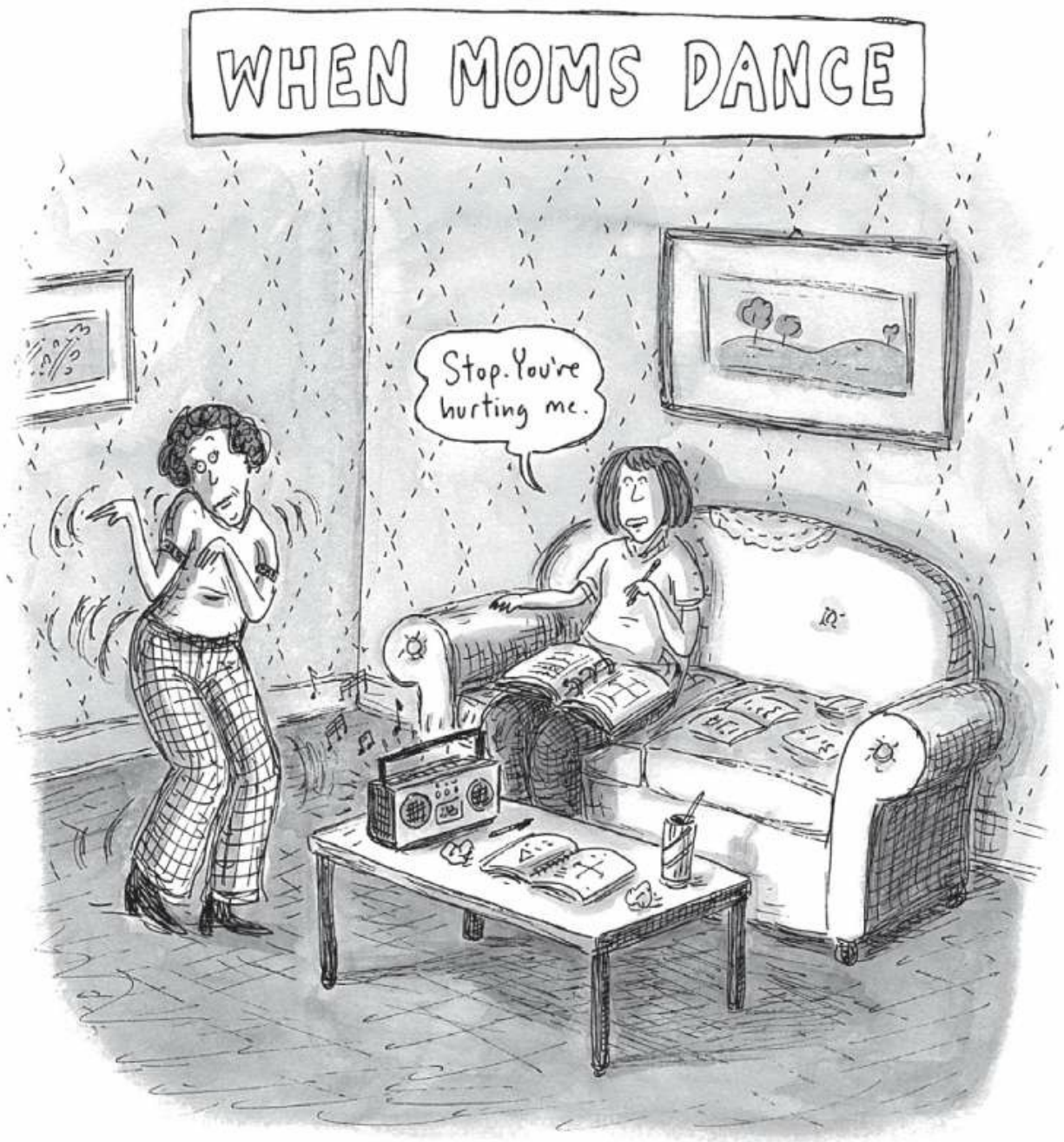
quickly at first and then gets slower (as is seen with memories for lists of nonsense words in [Figure 1.6](#) and for passive forgetting of television shows in [Figure 7.6A](#)).

Does the mere passage of time cause a skill to be “unlearned”? It often may seem this way, but forgetting can also result when new memories interfere with the recollection of old memories. As time passes, you perform more new skills, creating more memories that potentially interfere with the recollection of earlier skill memories. ([Chapter 7](#) notes that both interference and decay are also involved in the forgetting of memories for events and facts.) Much of this interference can occur without any awareness on the part of the person attempting to recall a skill. For instance, you might have difficulty recalling some of the dances you learned when you were younger but may easily recall dance steps you learned recently. Rather than thinking this recent learning is hampering your ability to perform the old dances, you would probably assume that you can’t remember an older dance simply because it has been so long since you last did it. However, there is no simple way for you to distinguish whether your forgetting is due to the passage of time or due to interference.

Interference between skill memories can occur even within a single day of learning a new skill. For example, in one series of studies, students were trained to perform a key-pressing task, similar to the serial reaction time task discussed earlier. This simple task qualifies as a perceptual-motor skill because the students’ ability to perform it improved with practice. Interestingly, the students demonstrated more rapid and accurate pressing times after a period of sleep ([Walker, Brakefield, Hobson, & Stickgold, 2003](#); [Walker, Brakefield, Morgan, Hobson, & Stickgold, 2002](#); [Walker, Brakefield, Seidman, et al., 2003](#)). If students learned to press keys in two different sequences on the same day, sleep-dependent enhancement of their performance was seen only for the *second* sequence learned. ([Chapter 7](#) considers the role of sleep in learning and memory.) If the participants learned the two sequences on separate days, then sleep enhanced their performance on both sequences. But if participants learned the first sequence on the first day, then on the second day reviewed that sequence immediately before learning the second sequence, subsequent sleep enhanced their accuracy on only the second sequence.

Thus, not only can practicing two skills on the same day interfere with retention of memories for the first skill, but reviewing a recently learned skill before beginning to practice a new one can interfere with subsequent recall of the skill that was reviewed! You might also have noticed in

this example that the condition that enhanced retention the most was the one with the most spacing between practice sessions. Once again, spacing appears to benefit retention.



Roz Chast The New Yorker Collection/The Cartoon Bank

Mom dancing: retrieval interference, skill decay, or lack of good judgment?

These findings highlight the intimate relationship between skill acquisition and skill recall and the fragile nature of newly acquired skill memories. Note, however, that athletes and musicians commonly practice multiple skills in parallel with no obvious evidence of interference, and variable practice generally leads to better long-term performance than constant practice. Thus, skills more complex or more distinctive than learning sequences of finger movements may be less susceptible to interference effects.

Interim Summary

- Memories for perceptual-motor skills are memories for sensory-guided movement patterns; they enable us to flexibly perform most physical actions.
- Memories for cognitive skills are memories for how to solve problems, apply strategies, and manipulate information; they enable us to think.
- Skill memories differ from memories for facts and events in that they are more difficult to verbalize, depend more on practice, and are often learned unconsciously.
- Feedback about performance, or knowledge of results, is critical to the effectiveness of practice in improving skills.
- The power law of practice, which holds for a wide range of cognitive and perceptual-motor skills, states that the extent to which practice leads to further improvement decreases with further practice.
- Several principles determine how new skill memories are formed and generalize: (1) massed practice produces better performance in the short term, but spaced practice leads to faster learning and better retention; (2) constant practice often does not improve performance or generalization as much as variable practice; (3) gradual increases in task difficulty can enhance skill learning more effectively than only difficult practice, despite requiring less effort.
- Practice can decrease the effects of past learning on performance and increase the effects of genetic influences.
- Generalization of skill learning depends on the similarity between the conditions experienced during retrieval and the conditions experienced while learning the skill.
- Repeatedly practicing similar skills can lead to the formation of learning sets that facilitate rapid acquisition of related skills.

8.2 Brain Substrates

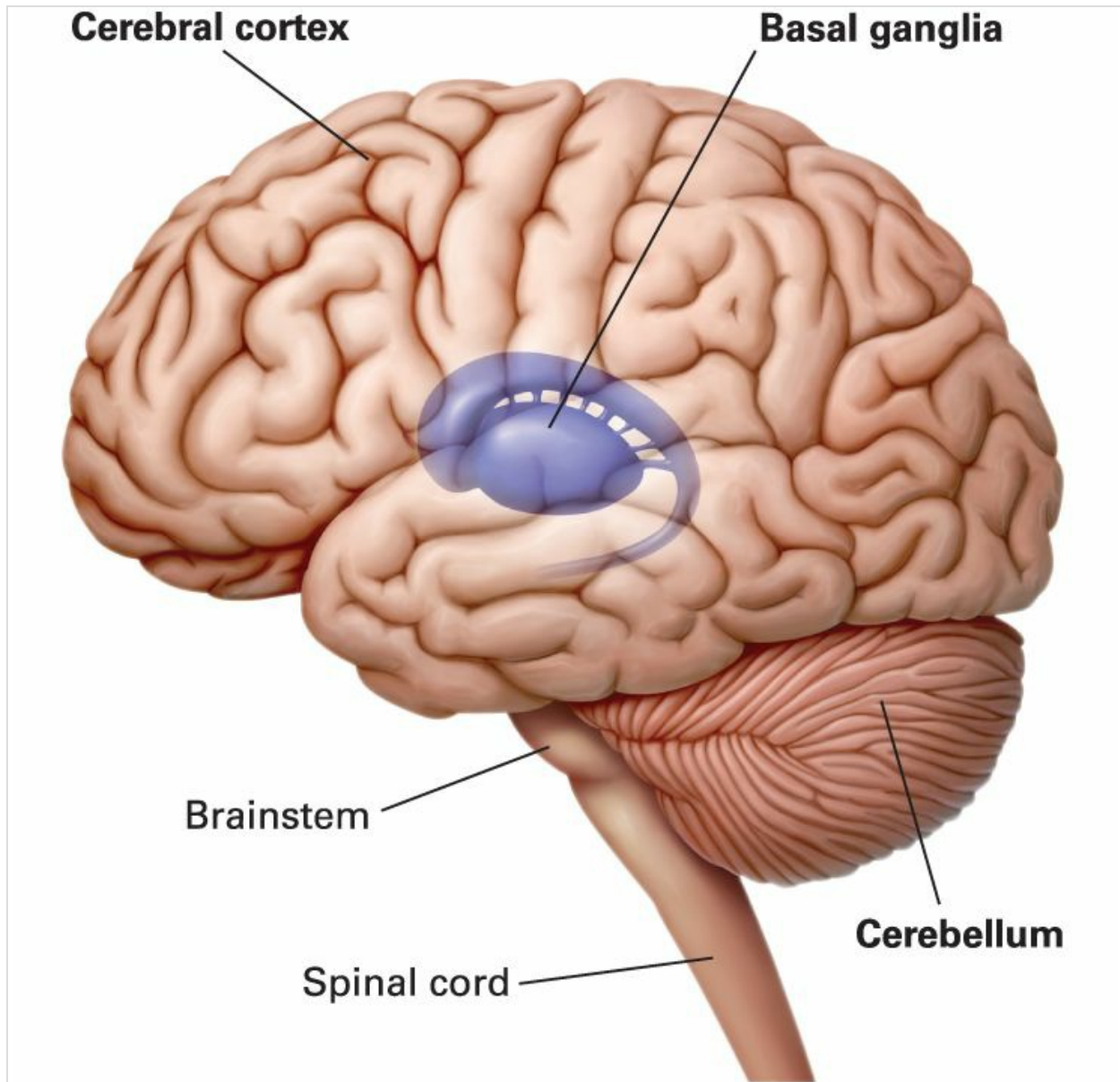
What neural systems do humans and other animals need in order to acquire memories of perceptual-motor and cognitive skills? Is there something special about the human brain that makes acquisition of new skills easier for humans than it is for other animals? Or do humans use the same neural systems to learn skills as other animals do, but in slightly different ways? How might we judge whether the skill memories that underlie a dolphin's ability to use a sponge differ from the skill memories a human uses to juggle?

Neuroscientists have used neuroimaging and neurophysiological recordings to monitor brain activity in humans and other animals during the acquisition and performance of skills. These techniques have enabled researchers to identify several brain regions involved in the formation and recall of skill memories. Scientists have also compared brain activity in experts and amateurs performing various skills, as well as in individuals before and after they have learned a particular skill. Neuropsychological studies of skill learning by patients with brain damage are also an important source of information. Through these kinds of research, scientists hope to associate stages of skill acquisition with changes in brain structure and activity.

All movements and postures require coordinated muscle activity. As discussed in [Chapter 2](#), a major function of the nervous system is to initiate and control patterns of muscle activity. The spinal cord and brainstem play a critical role in the performance of perceptual-motor skills by controlling and coordinating movements. Brain regions dedicated to sensation and perception, including the sensory cortices, are also involved, processing information that contributes to the guidance of movements. Of these, the somatosensory and visual systems (described in [Chapter 2](#)) are particularly important for learning perceptual-motor skills. Remember the experiment described earlier in this chapter, in which researchers instructed the participant to kick a target as quickly as possible? The kicker improved by processing visual feedback about how effectively he was coordinating the muscles in his leg.

In this section, we describe how practicing skills can change neural circuits. Although you can form skill memories in ways other than practice (e.g., by studying videos of expert athletes, by reading books about how to win at poker), neuroscientists have focused much of their effort on understanding the incremental effects of practice on brain activity during skill learning. The core elements of incremental skill learning seem to depend in particular on three brain regions

(introduced in [Chapter 2](#) and discussed in [Chapters 3](#) through [7](#)): the basal ganglia, the cerebral cortex, and the cerebellum ([Figure 8.6](#)). When any of these brain regions becomes dysfunctional, the performance and learning of both cognitive and perceptual-motor skills become seriously impaired.



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

FIGURE 8.6 Brain regions that contribute to skill learning Skill-memory systems in the brain include the basal ganglia, cerebral cortex, and cerebellum. These three regions modulate the control of movements by circuits in the brainstem and spinal cord.

Description

The labeled parts are as follows. Cerebral cortex lies in the upper part of the brain. Basal ganglia, a bulb-like structure is found deep within the cerebral hemispheres of the brain. The cerebellum lies in the hindbrain, adjacent to the spinal cord. The brain stem lies in the posterior part of the human brain and continues as the spinal cord.

The Basal Ganglia and Skill Learning

Basal ganglia is one of the few names for a brain structure that literally describe the region (or in this case, regions) to which they refer. The [basal ganglia](#) are a collection of *ganglia* (clusters of neurons) that lie at the *base* of the forebrain (the most prominent part of the human brain). They are positioned close to the hippocampus (see [Figure 2.3](#)), and, like the hippocampus, they receive large numbers of inputs from cortical neurons. In fact, most cortical areas send inputs to the basal ganglia. These inputs provide the basal ganglia with information about what is happening in the world—in particular, about the sensory stimuli a person is experiencing. Many of these cortical inputs are initially processed by the *dorsal striatum*, which is a subregion of the basal ganglia that plays an important role in operant conditioning (described in greater detail in [Chapter 5](#)). The basal ganglia send output signals mainly to the thalamus (affecting interactions between neurons in the thalamus and motor cortex) and to the brainstem (influencing signals sent to the spinal cord). By modulating these motor control circuits, the basal ganglia play a role in initiating and maintaining movement.

basal ganglia

A brain region that lies at the base of the forebrain.

The basal ganglia are particularly important for controlling the velocity, direction, and amplitude of movements, as well as for preparing the body to move ([Desmurget, Grafton, Vindras, Grea, & Turner, 2003](#); [Graybiel, 1995](#); [Turner, Grafton, Votaw, Delong, & Hoffman, 1998](#)). For example, suppose you are performing the rotary pursuit task. You need to move your arm in a circle at a velocity matching that of the rotating target. In this task, your basal ganglia will use information from your visual system about the movements of the target, the stylus, and your arm, as well as information from your somatosensory system about the position of your arm, to help control the direction and velocity of your arm movements. Similarly, if you dive into a pool to retrieve a coin, your basal ganglia will help you avoid colliding with the bottom of the pool.

Given all the interconnections between the basal ganglia and motor systems, it is not surprising that disruption of activity in the basal ganglia impairs skill learning. Such disruption does not, however, seem to affect the formation and recall of memories for events and facts. Consider the case of Muhammad Ali, one of the most agile and skilled boxers of his era; his career was ended by a gradual loss of motor control and coordination. Doctors identified these deficits as resulting from Parkinson's disease, a disorder that disables basal ganglia circuits (as discussed in more detail later in the chapter). Over time, the loss of basal ganglia function resulting from Parkinson's disease affects even the most basic of skills, such as walking. Whereas H.M.'s hippocampal damage (described in [Chapter 7](#)) prevented him from reporting on his past experiences, Ali's basal ganglia dysfunction hindered his use of skill memories and his ability to learn new skills; it did not affect his memory for facts or events.

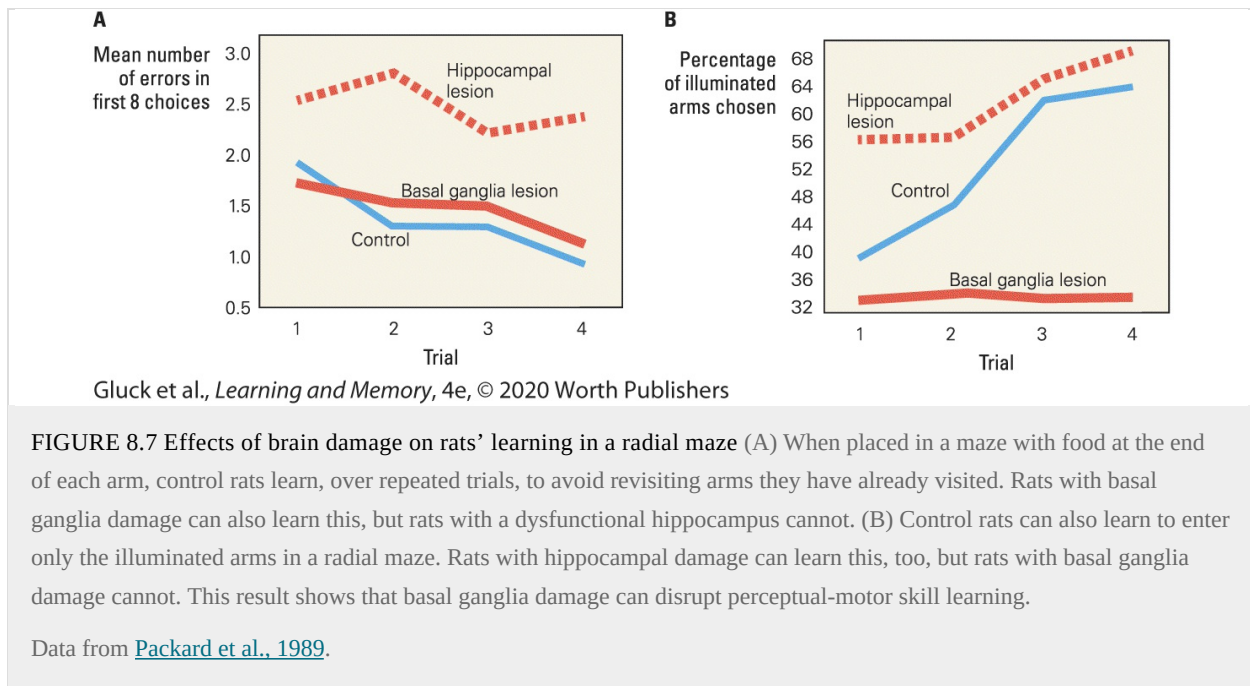
Many researchers suspect that the basal ganglia play a key role in forming skill memories, although the specific role is still debated ([Barnes, Kubota, Hu, Jin, & Graybiel, 2005](#); [Graybiel, 2005, 2008](#)). Most researchers agree, however, that practicing a skill can change how basal ganglia circuits participate in the performance of that skill and that synaptic plasticity is a basic neural mechanism enabling such changes ([Conn, Battaglia, Marino, & Nicoletti, 2005](#); [Graybiel, 2008](#)). We describe here experimental results that show the importance of the basal ganglia not only for performing skills but also for forming and accessing skill memories.

Learning Deficits After Lesions

Much of what is known about the role of basal ganglia in skill learning comes from studies of rats learning to navigate mazes, such as the radial maze shown in [Figure 7.2](#). In the standard radial maze task, rats learn to search the arms in the maze for food without repeating visits to the arms they have already searched. This task simulates some features of natural foraging (because food does not magically reappear at locations where a rat has just eaten) and has often been used to test rats' memories for events and locations (as discussed in [Chapter 7](#)). However, the entrances to the arms of the maze are all very similar, so unless the rat remembers specifically which arms it has visited, it is likely to reenter the same arm more than once.

In early sessions, this is just what rats do. They often go to the same arm multiple times and consequently waste a lot of time running back and forth along arms that contain no food. With practice, healthy rats (e.g., the control group in [Figure 8.7A](#)) learn that they can get more food

for their effort by keeping track of where they have been, and they make fewer errors of reentering previously visited arms. Their performance improves incrementally with practice, so their ability to navigate through the maze is a skill. Food acts as a kind of feedback in the radial maze task, in that correct performance leads to food.



Description

The graph in section A plots the mean number of errors in the first 8 choices on the vertical axis against trails on the horizontal axis. The readings on the vertical axis range from 0.5 to 3.0 in increments of 0.5, and the trials are numbered from 1 through 4. The graph shows three curves representing three groups of rats: hippocampal lesion, basal ganglia lesion, and the control group. The data in the graph is as follows.

Hippocampal lesion: trial 1-2.5, trial 2-2.75, trial 3-2.3, and trial 4-2.4.

Basal ganglia lesion: trial 1-1.75, trial 2-1.6, trial 3-1.5, and trial 4-1.3.

Control group: trial 1-2.0, trail 2-1.4, trial 3-1.5, and trial 4-1.0.

The graph in section B plots the percentage of illuminated arms chosen on the vertical axis against trails on the horizontal axis. The readings on the vertical axis range from 32 to 68 in increments of 4 and the trials are numbered from 1 through 4. The graph shows three curves representing three groups of rats: hippocampal lesion, basal ganglia lesion, and the control group. The data in the graph is as follows.

Hippocampal lesion: trial 1-56, trial 2-56, trial 3-64, and trial 4-68.

Basal ganglia lesion: trial 1-33, trial 2-34, trial 3-33, and trial 4-33.

Control group: trial 1-39, trail 2-46, trial 3-62, and trial 4-65.

All the above data is approximate.

To learn to navigate the radial maze efficiently, rats must remember certain aspects of past events. Not surprisingly, rats with hippocampal damage have major problems with this task ([Figure 8.7A](#)). Even after many sessions, they continue to visit arms they have visited before. In contrast, rats with basal ganglia damage learn this task as easily as rats with no brain damage. This shows that basal ganglia damage does not disrupt rats' memories for events, nor does it prevent them from performing the skills necessary to find food in a radial maze.

Researchers can modify the radial maze task slightly to make it less dependent on memories of past events. If, instead of putting food in all the arms, the experimenter places food only in arms that are illuminated, healthy rats quickly learn to avoid the non-illuminated arms (control group in [Figure 8.7B](#)). Rats with hippocampal damage can learn this version of the task because they only need to associate light with food, which does not require keeping track of arms they have visited. Yet rats with basal ganglia damage have difficulty learning this "simpler" version of the task. They continue to search non-illuminated arms even though they never find food in those arms ([Packard, Hirsh, & White, 1989](#)). Basal ganglia damage seems to prevent rats from learning the perceptual-motor skill of avoiding dark arms and entering illuminated arms.

Rats with brain damage show similar learning deficits in another task: the Morris water maze. In the standard version of this maze, experimenters fill a circular tank with murky water. They then place rats in the tank, and the rats must swim around until they discover a platform hidden just beneath the water surface. Once a rat finds the platform, it can climb out of the water, and the trial is over. Researchers measure the time it takes a rat to find the platform and use this as a measure of learning. Healthy rats gradually learn the location of the hidden platform after repeated trials in the tank. In this case, what the rats have learned is to direct their swimming movements based on visual cues present around the tank. With practice, rats become better at selecting which directions to swim, given their observations of which cues are currently visible. Rats with hippocampal damage have severe difficulties learning this standard task but have no problem learning the task if the platform is visible at the surface of the water. Rats with basal ganglia damage can learn to swim to the location of the platform whether it is visible or not. This seems to suggest that basal ganglia damage does not affect a rat's ability to learn this task.



Used with permission of Med Associates, Inc.

Which brain regions might this rat be employing to learn where a hidden platform is located?

Tests of transfer of training, however, tell a different story. If experimenters move a visible platform in the Morris water maze to a new location during testing, rats with hippocampal damage (or no damage) swim directly to the platform to escape the water. Rats with basal ganglia damage, on the other hand, swim first to where the platform *used* to be; only afterward do they find the platform in its new location ([McDonald & White, 1994](#)). One interpretation of this finding is that rats with basal ganglia damage have difficulty learning to swim toward a platform to escape the water (even when the platform is clearly visible) and instead learn to swim to a particular location in the tank to escape the water. This study illustrates that just because two animals may seem to be performing a skill in the same way does not mean that their skill memories and their ability to use those memories in novel situations are equivalent.

These and other experiments with rats have led researchers to conclude that the basal ganglia are particularly important in perceptual-motor learning that involves generating motor responses based on specific environmental cues. The basic assumption behind such research is that there is nothing unique about the way the basal ganglia function in rats learning to navigate mazes; consequently, basal ganglia damage should disrupt skill learning in similar ways in humans. Conversely, enhanced basal ganglia function may facilitate skill learning. For example,

researchers found that individuals with larger basal ganglia were the quickest to improve at the video game Space Fortress ([Erickson et al., 2010](#)). (See [“Learning and Memory in Everyday Life”](#) for more insight into how video game playing might in turn affect brain function.)

LEARNING AND MEMORY IN EVERYDAY LIFE

Are Video Games Good for the Brain?

Since the advent of television, people have been spending much of their lives staring at the glow of a rectangular screen. Video games have transformed passive viewing into an interactive experience, and today’s video games are as complex as any sport, card game, or board game. They are quickly replacing other recreational activities as the preferred pastime of children around the world. Many parents are concerned that this new pastime is turning children’s brains into mush and that the skills acquired by playing such games are worthless. What is actually going on? Does video game playing have a negative impact on a person’s mental capacities?

Video games have some advantages over traditional games. They offer a wide variety of experiences and options, they build expertise without requiring instruction from an expert, they present minimal risk of injury, and they can be played in any weather at any time. On the other hand, video games have been blamed for provoking teen violence, contributing to obesity and a general lack of physical fitness, reducing literacy, decreasing opportunities for face-to-face interactions with family members and peers, and occupying children’s minds with useless information (see [Anderson & Bushman, 2001](#), for an early review of the scientific literature on this topic).

To date, the question of whether video games are good or bad for the brain has received little scientific study. One series of experiments found that college students who played high-action video games (e.g., Grand Theft Auto 3, Crazy Taxi, Counter-Strike, and Spider-Man) at least 1 hour a day, at least 4 days a week, for at least 6 months had increased visual attention abilities compared with students who did not play video games ([Green & Bavelier, 2003](#)). The reported benefits of playing fast-action games included increased visual capacity and enhanced spatial attention, with an increased ability to apprehend and count sets of visual stimuli ([Bediou et al., 2018](#)). Interestingly, a control group that spent the same amount of time playing Tetris, a non-action video game, showed no enhancement in visual attention. However, critics suggest that the apparent benefits in these studies are a statistical artifact rather than an effect of video game playing ([Unsworth et al., 2015](#)).

There is increasing interest in using video games as educational aids ([Howard-Jones et al., 2014](#)) and as a way of slowing down cognitive decline in the elderly ([Mishra & Gazzaley, 2014](#)). Extensive playing of video games undoubtedly can change the structure and function of a player’s brain; if it didn’t, players would never improve. Do such brain changes have benefits (or costs) for any skills other than video game playing? As with transfer of other skills, the answer likely depends on how much overlap there is between the skills learned in the games and skills used in other, nongaming situations.

Neural Activity During Perceptual-Motor Skill Learning

Measures of neural activity in the basal ganglia during learning provide further clues about the role of the basal ganglia in the formation of skill memories. Experimenters can train rats to turn right or left in a T-shaped maze in response to a sound cue that the rats hear just before reaching

the intersection where they must turn ([Figure 8.8](#)). For example, an experimenter releases a rat in the maze, and then a computer plays a specific sound, instructing the rat to make a right turn. If the rat turns to the right, the experimenter gives the rat food (which, as discussed in [Chapter 5](#), is a particularly effective form of feedback). With practice, rats learn to perform this simple perceptual-motor skill accurately. Researchers can use electrodes implanted in rats before training to record how neurons in the basal ganglia fire as rats learn the T-maze task ([Jog, Kubota, Connolly, Hillegaart, & Graybiel, 1999](#)).

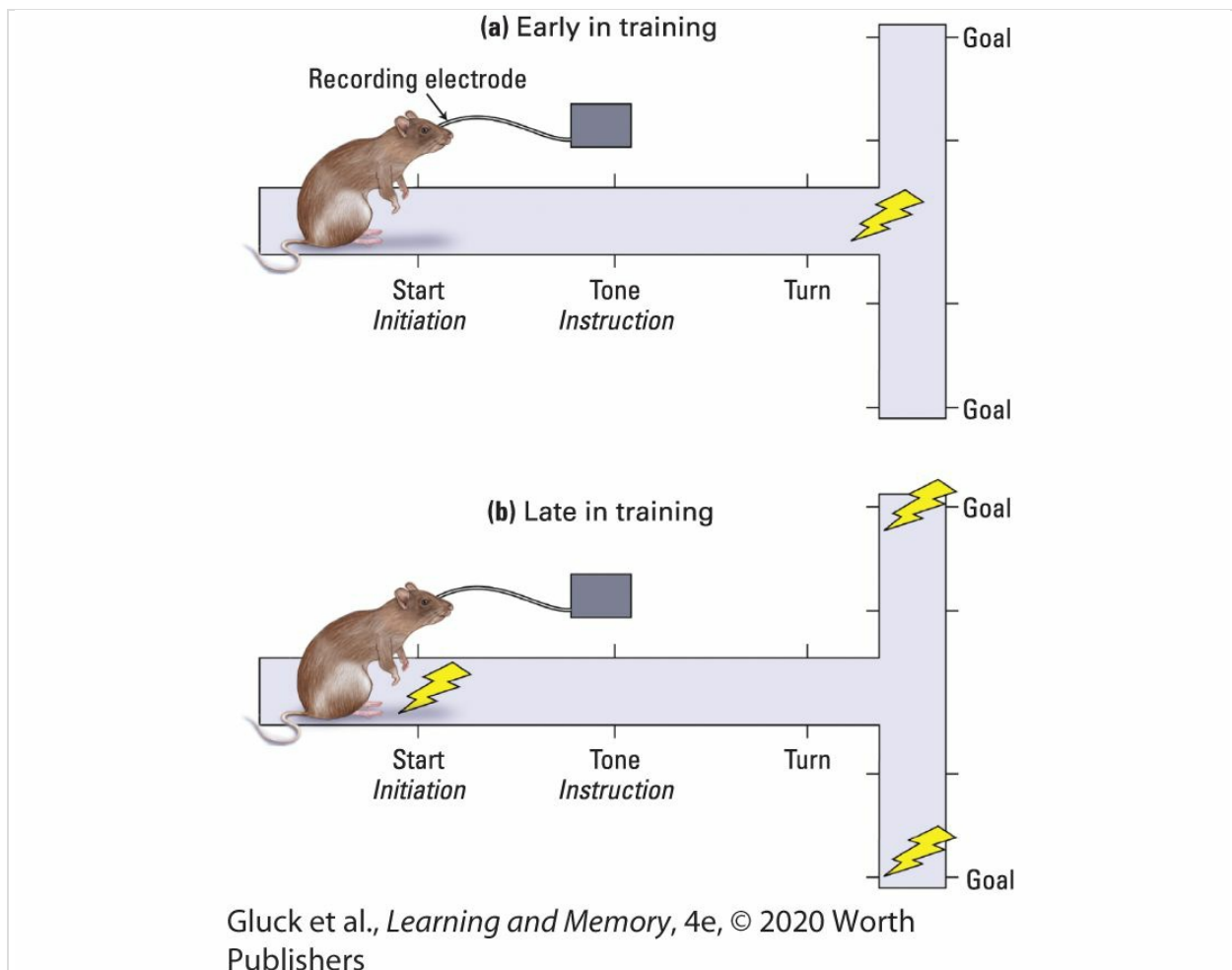


FIGURE 8.8 Changes in basal ganglia firing patterns during skill learning (A) Researchers implanted electrodes in rats' basal ganglia and then trained the rats to turn right or left in a T maze after hearing a tone instruction. Early in training, 50% of basal ganglia neurons fired strongly (indicated by the lightning bolt) when the rats chose which direction to turn. (B) As training progressed, basal ganglia neurons began to fire mainly at the beginning and end of the rats' movements through the maze; finally, more than 90% of neurons fired almost exclusively when rats were at these positions.

Research from [Jog et al., 1999](#).

Description

Early in training: A rat is at the start of a T-shaped maze. A recording electrode is attached to its head. Three phases are labeled on the horizontal limb of the T as follows. Start initiation, tone initiation, and turn. The vertical limb has two goals labeled at its upper and lower ends. A lightning bolt is shown at the intersection point of the maze.

Late in training: The three phases in the T shaped maze are labeled in the same pattern as in the early training stage. Three lightning bolts are shown one at the start initiation phase, and two at the goals.

Such recordings revealed four basic patterns of neural activity in the basal ganglia when rats were in the T maze: (1) some neurons fired most at the start of a trial, when the rat was first released into the maze; (2) some fired most when the instructional sound was broadcast; (3) some responded strongly when the rat turned right or left; and (4) some fired at the end of a trial, when the rat received food. During the early stages of learning, about half of the recorded basal ganglia neurons showed one of these four patterns of activity. *Most of these neurons fired only when a rat turned right or left in the maze* ([Figure 8.8A](#)). The other half of the recorded neurons fired in ways that were not clearly related to the rats' movements or experiences in the maze. As the rats' performance improved with practice, the percentage of neurons that showed task-related activity patterns increased to about 90%, *with most neurons firing strongly at the beginning and at the end of the task rather than during turning* ([Figure 8.8B](#)). These measurements show that neural activity in the basal ganglia changes during the learning of a perceptual-motor skill, suggesting that encoding or control of skills by the basal ganglia changes as learning progresses.

The increased neural activity seen in the beginning and end states during the maze task after the rats had become familiar with it suggests that the basal ganglia had now developed (or were now monitoring) a motor plan that was initiated at the beginning of each trial. The motor plan then directed the rat's movements until the trial ended ([Graybiel, 2008](#)). This hypothetical process is consistent with Fitts's model of skill learning, in which automatically engaged motor programs gradually replace active control of movements ([Fitts, 1964](#)). Someone learning to juggle might show similar changes in basal ganglia activity—that is, if we could record signals from her neurons, which is not yet possible. In a novice juggler who is still in the cognitive stage of skill acquisition, basal ganglia neurons might fire most strongly when the balls are in the air (when an action must be chosen based on visual information). In an expert juggler who has reached the autonomous stage of skill learning, basal ganglia neurons might fire most strongly when she is catching and tossing the balls.

The data presented here show that the basal ganglia contribute to learning of perceptual-motor skills. Do they also contribute to cognitive skill learning?

Brain Activity During Cognitive Skill Learning

Neuroimaging studies of the human brain reveal that the basal ganglia are indeed active when participants learn cognitive skills ([Poldrack et al., 2001](#); [Poldrack, Prabhakaran, Seger, & Gabrieli, 1999](#); [Seger & Cincotta, 2006](#)). In one series of experiments, participants learned to perform a classification task in which a computer presented them with sets of cards and then instructed them to guess what the weather would be, based on the patterns displayed on the cards ([Figure 8.9A](#)). Each card showed a unique pattern of colored shapes. Some patterns appeared when rain was likely, and others appeared when the weather was likely to be sunny. As each card was presented on-screen, participants predicted either good or bad (sunny or rainy) weather by pressing one of two keys. The computer then determined and reported the actual weather outcome based on the patterns on the cards. Participants had to learn through trial and error which patterns predicted which kind of weather ([Gluck, Shohamy, & Myers, 2002](#); [Knowlton, Squire, & Gluck, 1994](#)).

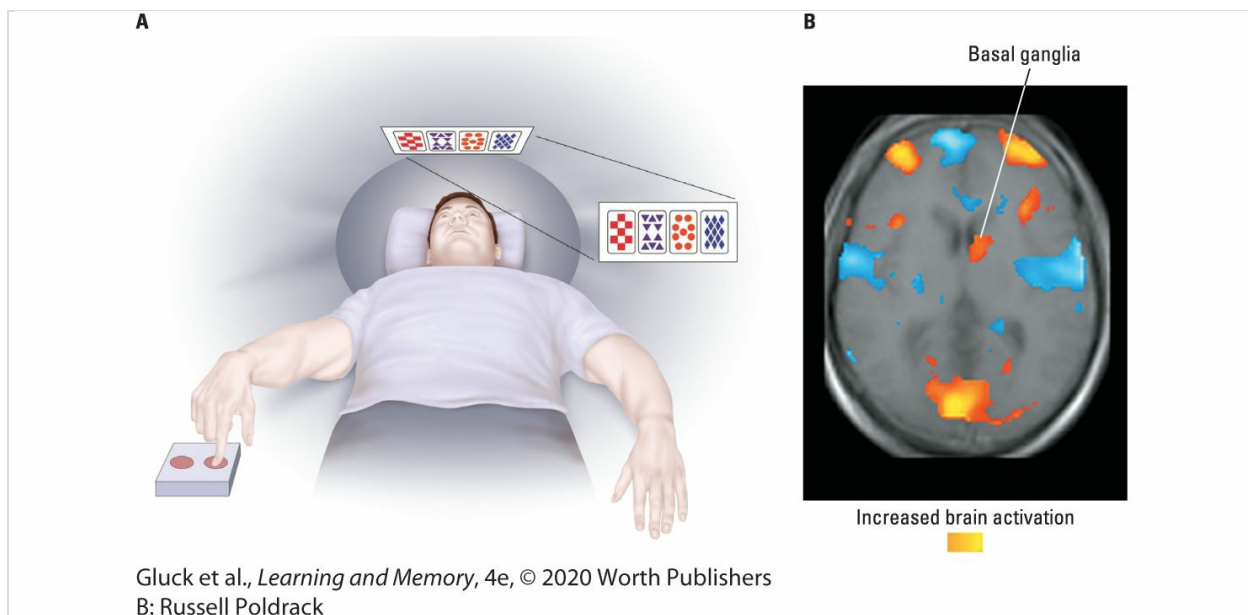


FIGURE 8.9 Neuroimaging during learning of the weather prediction task (A) A participant, lying with his head in the MRI scanner, is shown a set of cards on-screen that he must use to judge what weather conditions are likely to occur. Different patterns correspond to different predictions; for example, the pattern of squares on the leftmost card shown here predicts a 60% chance of rain. The participant is not given this information but must figure out through trial and error which patterns indicate a high chance of rain. (B) During the weather prediction task in a well-trained subject, fMRI difference

images show increased activation (orange) in the basal ganglia.

(B) Information from [Poldrack et al., 2001](#).

Description

The illustration shows a man lying on a scanning machine and pressing a button with his hands. A set of cards are displayed above his head. An extended view shows the same set of cards on the right side. Each card has a unique pattern imprinted on it. The fMRI imagery highlights the regions of increased brain activation on the posterior and the anterior parts of the brain and the basal ganglia (labeled).

The task mimics real-world weather prediction in that no combination of “patterns” (i.e., of cloud cover, temperature, wind, and so on) is 100% predictive of the weather that will follow; rather, meteorologists must develop a wide range of cognitive skills to accurately forecast the weather. For participants in this study, the task may have seemed more like reading tarot cards than learning a cognitive skill, but they usually did improve at it with practice.

Since each card was only associated with a likelihood that a particular kind of weather would occur, there was no simple rule that participants could use to make accurate predictions. Instead, to improve at the task, participants gradually had to learn which cards *tended* to predict certain types of weather. Brain-imaging data showed increased activity in the basal ganglia as individuals learned to make these judgments ([Figure 8.9B](#)). This and similar imaging studies suggest that the basal ganglia contribute to both cognitive and perceptual-motor-skill learning. But *how* do they contribute?

Despite considerable evidence that the basal ganglia enable skill learning, their specific function in this process is still debated. Because the basal ganglia are involved in the control and planning of movements, it is possible that damage to the basal ganglia leads to changes in movements that impair learning processes: if you can’t control how your arms are moving, you will have difficulty learning how to juggle. Consequently, changes in skill learning caused by lesions to the basal ganglia, as seen in rats learning the radial maze task, do not definitively prove that this region is critical for encoding or retrieving skill memories. By the same token, learning-dependent changes in the activity of basal ganglia neurons, as seen in rats learning to follow instructions in a T maze, could reflect changes in the information coming from the sensory cortex rather than changes generated by the basal ganglia.

Are basal ganglia neurons doing most of whatever is required to form memories of skills, or are other brain regions, such as the cortex and cerebellum, doing the bulk of the encoding and retrieval? Is it possible that the basal ganglia contribute no more to skill-memory formation than do other brain regions but that the basal ganglia are specialized for specific aspects of the learning process? We need to take a closer look at different cortical regions during and after practice sessions to shed light on these issues.

Cortical Representations of Skills

How important is the cerebral cortex for the learning and performance of skills? Given that most animals do not have a cerebral cortex and that animals born with a cortex can continue to perform many actions after surgical removal of all their cortical neurons, you might conclude that the cerebral cortex is not very important for either learning or performing skills. In fact, mammals are the only animals that make extensive use of cortical circuits for any purpose, so whatever the role of the cerebral cortex in skill memory, it probably plays this role most extensively in mammals. Coincidentally (or not), mammals are highly flexible, relative to most other species, when it comes to learning new skills.

Neural circuits in your cerebral cortex that become active when you dance, jump, or sing are continuously changing over time in ways that may enhance your ability to perform these actions. From this perspective, skill memories are the neural outcomes of repeated performances. A simple analogy is the way your muscles and performance change in response to a bodybuilding regimen. Just as increasing the strength and flexibility of your leg muscles can affect how well you jump, changing patterns of activity in your cortex through practice and performance may also influence your jumping ability.

Cortical Expansion

If cortical networks are like brain “muscles,” then you would expect that practicing different skills should affect different cortical regions, just as different physical exercises affect different muscle groups. Indeed, this seems to be true. Regions of the cerebral cortex involved in performing a particular skill often expand in area with practice, while regions that are less relevant to the skill show fewer changes. Neuroimaging techniques such as fMRI reveal this expansion by showing increased blood flow to particular regions. For example, brain-imaging studies of professional violinists showed that the cortical activation evoked in the somatosensory

cortex during use of the fingers that control note sequences was more extensive than in nonviolinists ([Elbert, Pantev, Wienbruch, Rockstroh, & Taub, 1995](#)). Interestingly, the cortical maps of violinists' bow hands (on which the fingers always move together) showed no such elaborations: the cortical changes were specific to the hand that moves the fingers separately. Similar differences in cortical activation have also been observed in the motor cortices of expert racquetball players when they use their playing hand versus their nonplaying hand ([Pearce, Thickbroom, Byrnes, & Mastaglia, 2000](#)).

Measures of blood flow reveal larger areas of activation in sensory and motor cortices after extensive practice, which implies that experience is affecting cortical circuits. These measures do not reveal what physical changes occur, however. Studies using structural MRI techniques indicate that practice can actually change the amount of cortical gray matter, which is where the cell bodies of neurons are found. For example, after about 3 months of training, people who learned to juggle three balls continuously for at least 1 minute showed a 3% increase in gray matter in areas of the visual cortex that respond to motion ([Draganski et al., 2004](#)). In fact, changes in gray matter can be observed as early as 7 days after training begins ([Driemeyer, Boyke, Gaser, Buchel, & May, 2008](#)). No comparable structural changes were observed in the motor cortex, basal ganglia, or cerebellum. It is not known whether this expansion of gray matter reflected changes in the number or size of synapses, changes in the number of glia (the cells providing functional and structural support to neurons), or changes in the size of cortical neurons.

Like neuroimaging studies in humans, electrophysiological studies in monkeys show that practice can expand cortical representations. In one such experiment, researchers trained monkeys to perform a tactile discrimination task ([Recanzone, Merzenich, Jenkins, Grajski, & Dinse, 1992](#)). The task required the monkey to release a handgrip whenever it felt a stimulus on its fingertip that differed from a standard stimulus. During each trial, the monkey initially felt a vibration having a certain fixed speed for about half a second on one of its fingers. This initial tactile stimulus, always the same, provided a standard for comparison. The initial stimulus was followed by a half-second interval of no stimulation and then a series of one to four additional vibrating stimuli, each vibrating either at the same rate as the standard or faster. The monkey was given fruit juice if it released the handgrip when vibrations were faster than the standard.

This task is similar to the T-maze task described earlier, in which researchers recorded the activity of basal ganglia neurons in a rat as it learned to turn right or left in response to acoustic cues. Both the T-maze and tactile discrimination tasks require the animal to perform one of two responses (in one task, turn right or turn left; in the other, grip or release) based on specific cues provided to a single sensory modality (sound in one task, touch in the other). These two tasks might not seem to require much skill compared with the musical performances of professional violinists or the physical feats of professional athletes, but it is important to keep in mind that the animals would never perform these trained actions in the wild. The criterion for whether an ability is a skill is not how hard it would be for a human to perform the action but whether the ability can be improved through experience.

When a monkey learned to respond to a vibrating tactile stimulus that predicted the delivery of juice, the area of the somatosensory cortex that processed the cue increased. As a result, monkeys that learned the tactile discrimination task had enlarged representations in the somatosensory cortex for the finger they used to inspect tactile stimuli. Studies such as this show that perceptual-motor skill learning is often associated with expansion of representations within the sensory cortex involved in performing the skill.

Similarly, practicing a perceptual-motor skill can cause representations within the motor cortex to expand. For example, electrical stimulation of the motor cortex (a technique introduced in [Chapter 2](#)) in monkeys trained to retrieve a small object showed expansion in the area of the cortex that controlled movements of the fingers ([Nudo, Milliken, Jenkins, & Merzenich, 1996](#)). Likewise, in monkeys that learned to turn a key with their forearm, representation of the forearm in the motor cortex expanded.

Researchers do not know how many different cortical regions are modified during learning of a particular skill, but the current assumption is that any cortical networks that contribute to performance of the skill are likely to be modified as training improves (or degrades) performance. Researchers also have yet to determine exactly how the expansion of cortical representations occurs, but most neuroscientists believe that representational expansion reflects the strengthening and weakening of connections within the cortex resulting from synaptic plasticity. (Basic mechanisms of synaptic plasticity are described in [Chapter 2](#).)

Although expansion of cortical regions is usually associated with superior performance, there is some evidence that too much expansion can cause problems. In a disorder called *musician's dystonia*, extensive practice playing an instrument can lead to a reduction or loss of motor control in the limbs or digits used to play rapid sequences. Experimental work indicates that this loss of control reflects excessive reorganization of the motor cortex ([Rosenkranz, Butler, Williamon, & Rothwell, 2009](#)).



Anatoli Styf/Shutterstock

Why might a professional violinist who practices daily suddenly start to lose the ability to correctly finger the strings when playing a familiar piece?

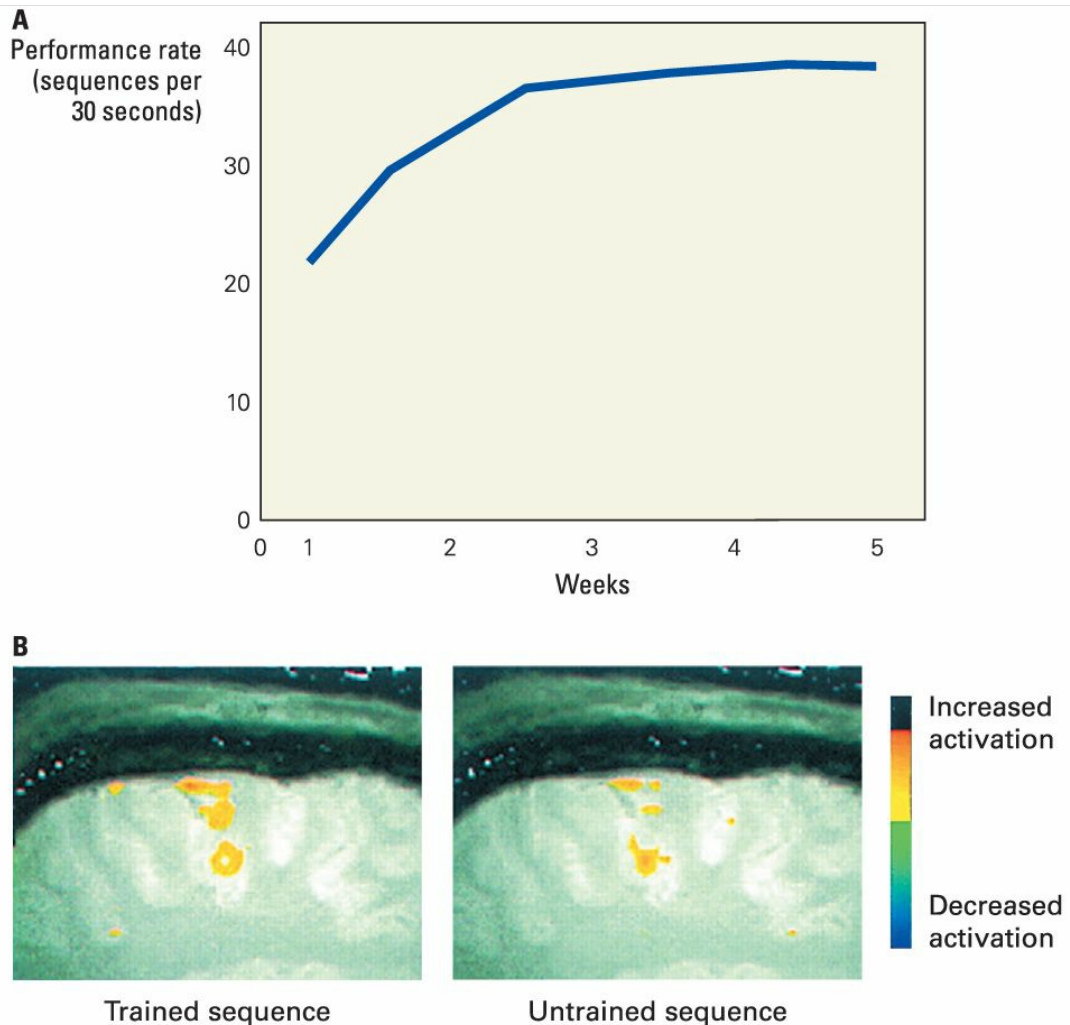
Are Skill Memories Stored in the Cerebral Cortex?

Many experiments have shown that cortical networks change as skill learning progresses, but this tells us only that the two phenomena are correlated, not that changes in the cerebral cortex actually cause the improved performance. Such studies also do not show for sure that skill memories are stored in cortical networks. As you saw earlier, changes in neural activity in the

basal ganglia also take place during skill learning. The cerebral cortex clearly contributes to skill learning and performance, but knowing this is not the same as knowing exactly what cortical circuits do during skill learning.

One way to get closer to understanding the cortical contribution to skill learning is to measure cortical activity during training. Much of what is known about skill learning pertains to how different practice regimens relate to differences in skill acquisition and forgetting; we saw this in [Section 8.1](#). If it were possible to show that cortical changes parallel known behavioral patterns or that improvements in performance can be predicted from cortical changes, we could be more certain that skill levels and cortical activity are closely related. Initial investigations in this direction suggest that the behavioral stages of skill acquisition are indeed paralleled by changes in cortical activity.

Data from brain-imaging studies show that when people begin learning a motor skill that requires sequential finger movements, the part of the motor cortex activated during performance of the task expands rapidly during the first training session but more gradually in later sessions. Avi Karni and colleagues required participants to touch each of their fingers to their thumb in a fixed sequence as rapidly and accurately as possible ([Karni et al., 1998](#)). In parallel with the changes seen in the motor cortex during training, participants' performance of this task improved rapidly in early sessions but more gradually in later sessions ([Figure 8.10A](#)), consistent with the power law of practice. Imaging data collected over 6 weeks of training suggested that practice beyond the third week of training (after performance had stabilized) resulted in additional increases in the representation of learned movements in the motor cortex. If performance had stabilized, why did the cortical representation continue to expand?



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

B: Karni, A. et al., "The acquisition of skilled motor performance: fast and slow experience-driven changes in primary motor cortex." PNAS 95 (1998): 861–8.
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FIGURE 8.10 Changes in skill performance and associated motor cortex during training (A) Participants who practiced performing a sequence of finger movements gradually increased the rate and accuracy with which they could perform this skill. The plot shows average scores for the group of participants. (B) After training, fMRI scans revealed that the area of motor cortex activated when participants performed the practiced sequence expanded (left panel) relative to the region activated when they performed an untrained sequence of identical finger movements (right panel).

Information from [Karni et al., 1998](#).

Description

The line graph plots performance rate in sequences per 30 seconds on the vertical axis against weeks on the horizontal axis. The readings on the vertical axis range from 0 to 40 in increments of 10 and the weeks are numbered from 1 through 5. The graph shows a line that passes through these points: Week 1: 22 sequences, Week 2: 33 sequences, Week 3: 37 sequences, Week 4: 38 sequences, Week 5: 38 sequences.

The f MRIs imageries depict brain activity of a trained sequence and an untrained sequence. The image corresponding to a trained sequence shows more brain activity in the motor cortex than untrained sequence. A legend on the side shows the colors corresponding to increased and decreased brain activation.

Overall, the region of motor cortex activated during performance of the practiced sequence expanded relative to the area activated by different, untrained sequences of finger movements ([Figure 8.10B](#)). Karni and colleagues hypothesized that the period of “fast learning” involves processes that select and establish the optimal plans for performing a particular task, whereas the subsequent slower stages of learning reflect long-term structural changes of basic motor control circuits in the cortex. Data from studies of perceptual-motor skill learning in rats are consistent with this interpretation. Rats trained in a reaching task showed significant differences in their motor map only after practicing the task for at least 10 days ([Kleim et al., 2004](#)). This finding suggests that structural changes in the cortex reflect the enhancement of skill memories during later stages of training.

A wide variety of sensory and motor events activate circuits in the cerebral cortex, so it is not surprising that these brain regions contribute to skill learning and performance. However, the respective roles of the cortex and basal ganglia in forming and recalling skill memories remain to be assessed and will require studying the interactions between the cerebral cortex and the basal ganglia while individuals are learning various perceptual-motor and cognitive skills.

TEST YOUR KNOWLEDGE

Which Cortical Regions Change During Skill Learning?

As skill learning progresses, cortical regions change in ways that mimic patterns of skill acquisition. Based on what is known from neurophysiological experiments with monkeys and from behavioral experiments with humans, name a cortical region that you think might show different activation levels in a chess grand master as compared with a beginner. ([Answers appear at the end of the chapter.](#))

The Cerebellum and Timing

What about skill learning in animals such as birds and fish, which do not have much cortex? Researchers can train pigeons to perform a wide range of perceptual-motor skills, and fish can rapidly learn to navigate mazes. Because they have little or no cortex, animals like these must rely on evolutionarily older parts of the brain to learn skills. One region that seems to be

particularly important in this process is the cerebellum. [Chapter 4](#) describes the key role the cerebellum plays in learning to predict important events, especially in the formation, execution, and timing of conditioned responses. These functions are also important for skill learning.

The cerebellum is probably one of the most basic neural systems involved in encoding and retrieving skill memories. Even an animal as lowly as a fish or frog, which may seem to have little potential for skill learning, has a cerebellum. And although you are not likely to see a fish or a frog performing in a circus, this does not mean these animals can't learn perceptual-motor skills; for example, with practice, fish can learn to press little levers for food. You are more likely to have heard parrots producing intelligible sentences or to have seen them riding tricycles. Birds, too, have cerebella, of course, which may facilitate their ability to learn such tricks. In fact, most animals that have a spine also have a cerebellum. Yet there are relatively few studies of cerebellar function in nonmammals. Consequently, much less is known about how the cerebellum contributes to skill-memory formation in animals that possess little cortex than is known about cerebellar function in mammals with lots of cortex.



Ma Jian/UPPA/ZUMA Press/Newscom

What might be the contributions of the cerebellum to the performance of this perceptual-motor skill?

Most of the inputs to the cerebellum are from the spinal cord, sensory systems, or cerebral

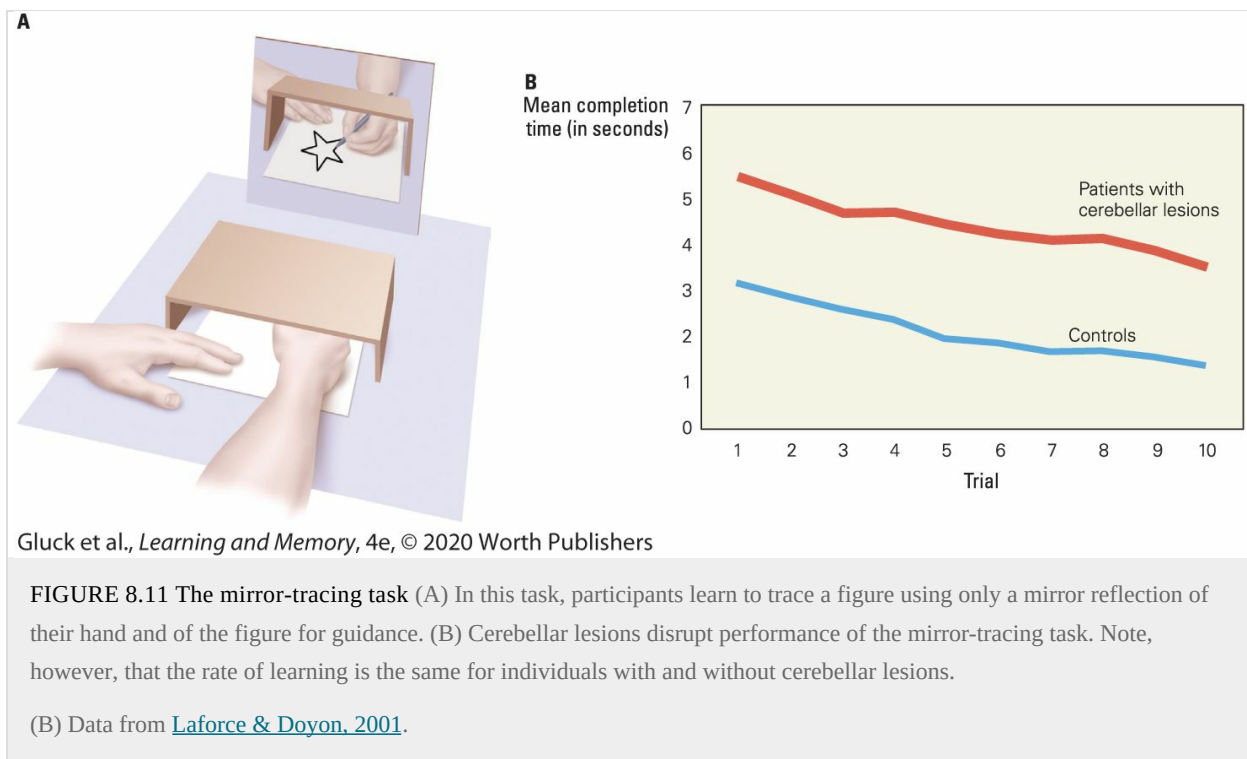
cortex, and most of the outputs from the cerebellum feed into the spinal cord or to motor systems in the cerebral cortex. Experiments conducted in the early 1800s showed that cerebellar lesions impair the performance of motor sequences. People with cerebellar damage, for example, have difficulty playing a musical instrument or writing. Collectively, these anatomical and neuropsychological data indicate that the cerebellum contributes to the performance of perceptual-motor skills in mammals. Because the structure of the cerebellum is organized similarly across different species, it is presumed to serve similar functions in both mammals and nonmammals ([Lalonde & Botez, 1990](#)).

Other evidence suggests that, in addition to facilitating the performance of skills, the cerebellum is involved in forming memories for skills. The cerebellum appears to be especially important for learning movement sequences that require precise timing, such as acrobatics, dancing, or competitive team sports. A person with cerebellar damage might be able to learn new dance moves but would probably have trouble learning to synchronize those moves to musical rhythms. For instance, early brain-imaging studies of systems involved in motor learning showed that there is a sudden increase in cerebellar activity when humans begin learning to perform sequences of finger movements ([Friston, Frith, Passingham, Liddle, & Frackowiak, 1992](#)). Similarly, rats that learn complex motor skills to navigate an obstacle course (e.g., balancing on tightropes and seesaws) develop predictable physiological changes in cerebellar neural circuitry, such as increased numbers of synapses ([Kleim et al., 1997](#)).

Cerebellar changes in acrobatic rats seem to depend on skill learning rather than on activity levels because rats that run in an exercise wheel for the same amount of time do not show such changes. Furthermore, studies have found that rats depend on cerebellar processing even when they learn to perform a task by simply watching other rats perform the task ([Leggio et al., 2000](#)). Rats that received cerebellar lesions after observing other rats navigate a maze benefited from those observations when they were later trained in the maze, whereas rats that received lesions before observing other rats traverse the maze did not benefit from their observations. Studies in humans have similarly revealed cerebellar involvement during observational learning of a perceptual-motor skill ([Torriero et al., 2011](#)), suggesting that memories of skills either performed or witnessed depend at least in part on cerebellar processing.

The cerebellum is also important for tasks that involve aiming at or tracking a target. To assess

such abilities, psychologists commonly use **mirror tracing**, a task in which individuals learn to trace drawings by looking in a mirror to observe their hand and the figure to be traced, which are otherwise hidden from view (**Figure 8.11A**). It is hard to draw well under these conditions, but if the cerebellum is working properly, the participant can eventually learn to perform this task well. In contrast, a person with cerebellar damage would find this task to be quite difficult. It took patients with cerebellar damage twice as long as individuals without damage to trace images shown in a mirror (**Figure 8.11B**), even after several sessions of training (**Laforce & Doyon, 2001**).



Description

A close-up view of a person's hand is shown writing on a sheet of paper underneath a wooden frame. Opposite to it, a mirror shows the close-up view of a hand, drawing a star.

The graph is labeled trial on the horizontal axis, with mean completion in time (in seconds) on the vertical axis. The readings on the vertical- axis range from 0 to 7 in increments of 1 and the trials on the horizontal axis are numbered from 1 to 10. The graph shows two descending curves representing patients with cerebellar lesions and controls. The curve representing patients with cerebellar lesions shows greater mean completion time than the patients in the control group for the same number of trials. The mean completion time decreases with an increase in trials for both curves.

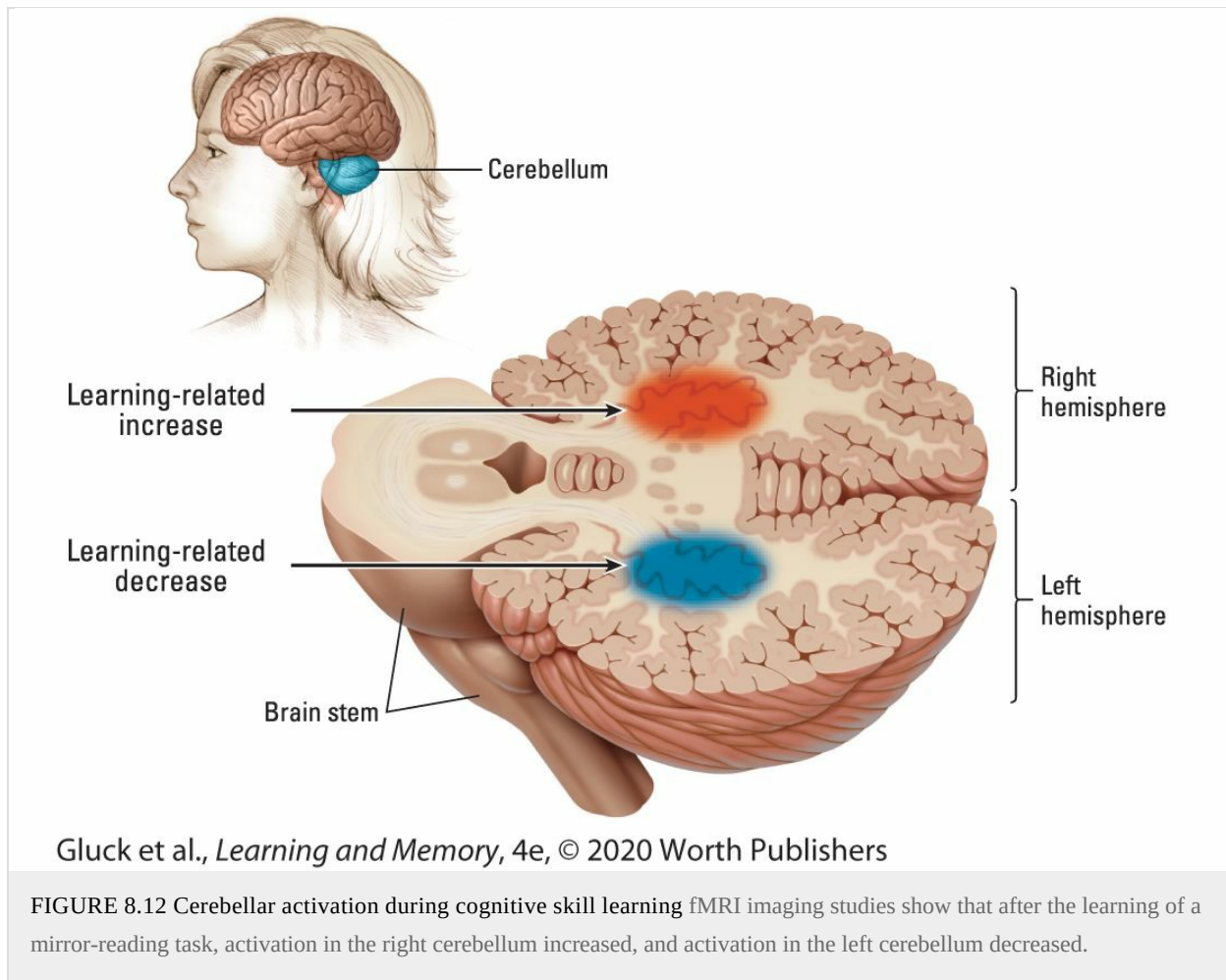
mirror tracing

An experimental task that requires individuals to trace drawings by watching a mirror image of their hand and of the figure to be

traced, with the actual hand and figure concealed; used to test perceptual-motor skill learning.

It is interesting to note in [Figure 8.11B](#) that the *rate* of learning for patients with cerebellar damage was comparable to that of the control group. This seems to suggest that learning in the patients with cerebellar damage was similar to that of the control group and that the patients simply performed more poorly. However, subsequent transfer tests in which both groups traced more complex figures revealed that the individuals in the control group benefited more from their training experiences than did the individuals with cerebellar damage. Thus, although the groups were learning at a similar rate, they were not transferring the mirror-tracing skill in the same way. This finding is similar to that in the study described earlier, in which rats with basal ganglia lesions seemed to have learned to find a visible platform in the Morris water maze in the same way as control rats but then performed differently when the platform was moved to a new location.

So far, we have discussed how the cerebellum contributes to perceptual-motor skill learning. Brain-imaging studies show that activity in the cerebellum also changes when individuals learn certain cognitive skills, such as [mirror reading](#). In the mirror-reading task, individuals learn to read mirror-reversed text. Researchers found that cerebellar changes that occur during learning of the mirror-reading task are *lateralized*—that is, they are different in each hemisphere ([Figure 8.12](#)): the left cerebellum shows decreased activity, whereas the right cerebellum shows increased activity with training ([Poldrack & Gabrieli, 2001](#)). Were you assuming that both sides of your brain are doing the same thing while you are reading this chapter? *Think again*. How such hemisphere-specific differences in cerebellar processing contribute to skill learning or performance is not yet known.



Description

A schematic of a woman is shown with the cerebellum highlighted within her brain, behind the brainstem, in the posterior ventral part of the brain.

A sectional view of the human brain is shown below. It is divided into two regions - Right hemisphere and Left hemisphere. A part of the cerebellum in the right hemisphere is highlighted and labeled Learning-related increase. A part of the cerebellum in the left hemisphere is highlighted and labeled learning-related decrease. The Brain Stem is labeled in the hindbrain.

mirror reading

An experimental task that requires individuals to read mirror-reversed text; used to test cognitive skill learning.

Keep in mind that almost all cognitive skills require the performance of some perceptually guided movements, if only eye movements. For example, we noted earlier in this chapter that chess masters move their eyes more efficiently to scan a chessboard than do less experienced players. Similar perceptual-motor skills may also be important for tasks such as mirror reading.

So it is possible that changes in cerebellar activity during the learning of cognitive skills might partially reflect the learning of motor sequences required for performing the cognitive activity.

In summary, then, the cerebellum, cerebral cortex, and basal ganglia are each critical to skill learning but in different ways. And yet, if you are having trouble learning a skill, there is no cut-and-dried division of labor between these three regions that would allow you to identify which part of your brain is to blame. The importance of each region for encoding or performing any given skill probably depends on the particular skill and your level of expertise. Nevertheless, the cerebellum seems most critical for timing; the cerebral cortex most critical for controlling complex action sequences; and the basal ganglia most critical for linking sensory events to responses. Knowing this, which brain region do you think would be most important for learning to run down stairs? The answer is probably all three—at different points in the learning process ([Hubert et al., 2007](#); [Steele & Penhune, 2010](#); [Yin et al., 2009](#)). Early on, the cerebellum, visual cortex, and motor cortex may work together to coordinate the timing and sequencing of leg movements. After extensive practice, the basal ganglia may begin to initiate and control more automatic sequences of leg movements. How these three brain regions work together during the acquisition and retention of skill memories is a question that researchers are still attempting to answer.

One feature that all three systems have in common is that skill learning is associated with gradual changes in the firing of neurons in these areas during performance of the skill. This finding means that practice can change the structure of neural circuits to make the control and coordination of movements (or thoughts, in the case of cognitive skills) more accurate and efficient. The most likely mechanism for such changes is synaptic plasticity. Understanding how and when the brain is able to adjust specific synapses within and between the cerebellum, basal ganglia, and cortex will clarify how humans and other animals learn skills.

Interim Summary

- Skill learning depends on three brain areas: the basal ganglia, the cerebral cortex, and the cerebellum.
- Studies of how rats with basal ganglia damage learn to navigate mazes suggest that the basal ganglia are critical for learning to generate motor responses based on environmental cues.

- Neural response patterns in the basal ganglia change during the learning of a perceptual-motor skill, suggesting that representations of that skill are dynamically modified as learning proceeds. The basal ganglia are also activated when people learn cognitive skills such as the weather prediction task.
- Regions of the somatosensory cortex and motor cortex needed to perform a particular skill expand with practice, but regions that are less relevant show fewer, if any, changes.
- The cerebellum is especially critical for learning and performing movement sequences that require precise timing (e.g., dancing) and tasks that involve tracking a target (e.g., mirror tracing).
- The cerebral cortex is mainly involved in controlling complex actions, and the basal ganglia link sensory events to responses.

8.3 Clinical Perspectives

[Chapter 7](#) describes how damage to the hippocampus and surrounding brain regions can disrupt memories for events and facts. Although there are no reports of brain damage that leads to a similarly dramatic *loss* of the ability to form skill memories, dysfunctions of the basal ganglia resulting from injury or disease can nonetheless interfere with the formation and use of skill memories. In addition, injuries to other parts of the brain or to the spinal cord or limbs can severely affect what a person can do. Patients often must make use of their ability to learn new skills to compensate. In this section, we explore some deficits caused by dysfunction in the basal ganglia, as well as new technologies that can help patients counteract disease- and injury-related impairments in skill performance.

Parkinson's Disease

[Parkinson's disease](#) is a nervous system disease involving disruptions in the normal functions of the basal ganglia and progressive deterioration of motor control ([DeLong & Wichmann, 2007](#); [Redgrave et al., 2010](#)). The main brain damage associated with Parkinson's disease is a reduction in the number of dopaminergic neurons in the substantia nigra pars compacta (SNc) that control activity in the basal ganglia. (See the discussion of the dorsal striatum in [Chapter 5](#).) Neurons in the SNc normally determine the levels of dopamine in the basal ganglia; when these neurons are gone, dopamine levels are greatly reduced.

Parkinson's disease

A disorder resulting from disruptions in the normal functioning of the basal ganglia and involving progressive deterioration of motor control and perceptual-motor skill learning.

Patients with Parkinson's disease show increasing muscular rigidity and muscle tremors and are generally impaired at initiating movements. Symptoms of the disease usually do not appear until after age 50 but can arise much earlier. People with Parkinson's disease find it harder to learn certain perceptual-motor tasks, such as the serial reaction time task and tracking tasks (including the rotary pursuit task); they also show deficits in learning some cognitive skills (e.g., predicting the weather from abstract shapes). Parkinson's disease may selectively disrupt circuits in the basal ganglia that contribute to the learning and performance of closed skills, especially those that have reached the autonomous stage of learning, where movements normally would be automatic ([Redgrave et al., 2010](#)).

Currently, the main treatments for Parkinson's disease are drug therapies to counteract the reduced levels of dopamine and surgical procedures to counteract the disruption caused by lack of dopamine in the basal ganglia. One surgical technique, [deep brain stimulation](#), has become the most effective neurosurgical technique for treating Parkinson's, but scientists are still not sure why it works ([Benabid, 2003](#); [Naskar, Sood, Goyal, & Dhara, 2010](#)). In deep brain stimulation, an electrical current is delivered through one or more electrodes permanently implanted deep in the patient's brain. Neurosurgeons place the ends of the electrodes near neurons that connect basal ganglia neurons with cortical circuits (e.g., in the thalamus or basal ganglia), as shown in [Figure 8.13](#). When electrical current from an implanted stimulator passes through these electrodes, many of the motor symptoms associated with Parkinson's disease, such as tremors, disappear within seconds, although they eventually return, and the stimulation must be repeated.

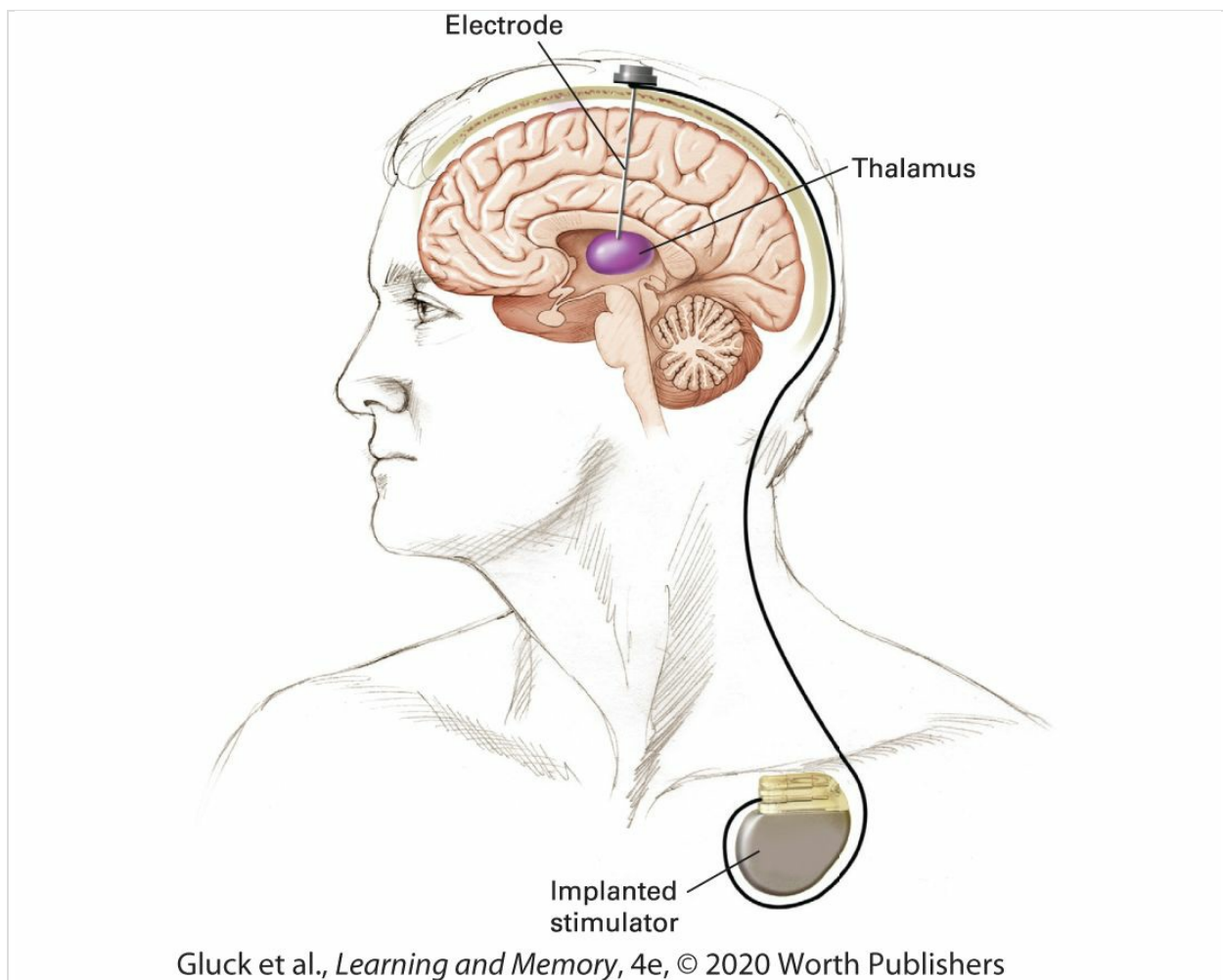


FIGURE 8.13 Deep brain stimulation for treatment of Parkinson's disease Neurosurgeons position the tip of an electrode

in a brain location (e.g., the thalamus) that, on stimulation, will affect neural activity in the basal ganglia and cerebral cortex. An implanted stimulator periodically passes current through this electrode to temporarily relieve symptoms of Parkinson's disease.

Description

A schematic shows a head-shot sketch of a human with the structure of the brain highlighted within his head. At the center of the brain, the thalamus is labeled and connected to an electrode fixed at the top of the head. The electrode is wired to an implanted stimulator, near the patient's clavicle.

deep brain stimulation

A procedure that involves delivering an electrical current into a patient's brain through one or more implanted electrodes; used to alleviate tremors and other motor symptoms associated with Parkinson's disease.

One idea about how this technique works is that without proper levels of dopamine, interactions between neurons in the cerebral cortex and the basal ganglia become locked into fixed patterns ([Dowsey-Limousin & Pollak, 2001](#)). This creates a situation similar to the endless back and forth of young children arguing (Child 1: "No, you be quiet!" Child 2: "No, you be quiet!" Child 1: "No, you ..."—ad infinitum) and disrupts the control of movements. Stimulation from the electrode is thought to quiet both brain regions, allowing normal brain activity to resume. Another possibility is that deep brain stimulation replaces disruptive activity in the basal ganglia and cerebral cortex with a new pattern of activity that is less disruptive ([Naskar et al., 2010](#)). Advances in understanding the brain systems that underlie skill memories will help doctors improve treatments like electrical stimulation for Parkinson's disease and use them more effectively.

Human–Machine Interfaces: Learning to Consciously Control Artificial Limbs

People who are limited in their ability to see or hear can take advantage of ever-evolving new electronic technologies to partially regain function in these sensory modalities (discussed in [Chapter 3](#)). Just as such *sensory prostheses* can help people overcome deficits in perceptual abilities, electromechanical devices called [motor prostheses](#) are being developed to help people recover lost abilities to learn and perform perceptual-motor skills. Unlike sensory prostheses, which automatically respond to environmental inputs (lights and sounds), motor prostheses must be consciously controlled by their users.

motor prosthesis

An electromechanical device that can help a person recover lost abilities to learn and perform perceptual-motor skills.

Like deep brain stimulation, use of motor prostheses requires a surgical procedure to implant electrodes in a person's nervous system. Instead of delivering electricity to neurons, however, these electrodes collect electrical signals from neurons and transmit those signals to a computer for processing. The computer then uses the signals to determine how to control the movements of a robotic limb. Generally, these limbs are artificial arms or hands, but they could be almost any appendage. Essentially, the computer transforms neural signals into robotic movements in real time.



UPMC

In what brain region would electrodes likely be implanted to enable a person to consciously control a robotic hand or arm to feed herself chocolate?

Much of the research on motor prostheses has been conducted on nonhumans, specifically monkeys and rats ([Borton, Micera, Millan, & Courtine, 2013](#); [Carmena et al., 2003](#); [Koralek, Jin, Long, Costa, & Carmena, 2012](#); [Velliste, Perel, Spalding, Whitford, & Schwartz, 2008](#)). Animals can be trained to control robotic limbs even if all of their motor abilities are intact. Researchers first develop programs that translate neural activity into signals that make a robotic arm move in

ways that match how, say, a monkey is actually moving its arm; then they restrain the monkey's arm and train the monkey to use the robotic arm to perform the same tasks ([Carmena, 2013](#)). This step involves transfer of training in that the monkey must retrieve its memories of the skills that it would normally use in the tasks and apply them to move the robotic arm. By reinforcing the monkey for performing tasks with the robot arm, researchers facilitate the transfer, and eventually the monkey acquires competent mind control of the robot arm ([Lebedev & Nicolelis, 2006](#)).

Some patients who have lost the ability to move any of their limbs, a condition called *tetraplegia*, have agreed to have similar electrodes implanted in their brains. Recordings from multiple electrodes positioned within the motor cortex have made it possible for these patients to control the movements of such devices as a mouse pointer on a computer screen, a motorized wheelchair, and a robotic arm that can grasp objects ([Collinger et al., 2013](#); [Hochberg et al., 2012](#)).

Motor prosthetics are also currently being tested for use in people who are missing arms or hands. In these cases, the electrodes are typically attached to nerves of the person's peripheral nervous system rather than implanted in the brain. In some cases, separate electrodes have been connected to incoming sensory pathways and outgoing motor pathways, making it possible for patients to both control the movements of an artificial hand or other prosthetic and to feel the consequences of those movements ([Talbot, 2014](#)). (See [Figure 2.9](#) for a reminder of how the incoming sensory pathways and outgoing motor pathways control movements.)

It might seem that a person who still possesses memories of how to perform basic perceptual-motor skills should be able to immediately use those memories to control similar movements with an artificial limb. Remember, however, that millions of neurons in multiple brain regions contribute to the performance of such skills. The electrodes that control the movements of robotic arms or prosthetic limbs are usually connected to relatively few neurons in a single part of the nervous system. Patients have to learn to generate neural activity that will enable the computer to determine what actions to initiate, much as Jedi in the *Star Wars* movies learned to use "the Force" to move objects with their thoughts. Like those who receive sensory prosthetics, humans and nonhumans with motor prosthetics gradually improve their ability to control them with practice ([Ganguly & Carmena, 2010](#); [Koralek et al., 2012](#); [Shenoy & Carmena, 2013](#)).

It remains to be seen how expert a human can become at mentally controlling artificial limbs or electronic devices. What is certain is that a better understanding of the mechanisms that enable humans to master new skills will be critical to maximizing people's ability to flexibly use such technologies.

Interim Summary

- Parkinson's disease involves disruptions in the normal functioning of the basal ganglia and progressive deterioration of motor control and skill-learning abilities.
- Deep brain stimulation can decrease the negative consequences of Parkinson's disease for skill performance by changing activity in the basal ganglia and cerebral cortex.
- Motor prostheses enable individuals with lost motor function or missing limbs to learn new ways of performing perceptual-motor skills.

Synthesis

Learning to dance with a partner in a social situation requires both perceptual-motor and cognitive skills, acquired and improved through observation and practice. Differentiating the cognitive aspects from the perceptual-motor ones can be difficult, as this chapter shows. Cognitive skills often depend on perceptual-motor skills (and vice versa) and may even become transformed into perceptual-motor skills over time.

Certainly, one cognitive aspect of dancing with a partner is the use of social skills to motivate someone to want to dance with you. Once you solve this problem—which in some cases may be as strategically challenging as a chess game—you face the perceptual-motor challenge of coordinating your own body movements with those of the other person, based on what you perceive of that person's maneuvers. Your skills at this point will depend on how much and how often you have practiced, as well as on the types of feedback you have received from past partners. Perhaps you are in the cognitive stage of learning to dance, still thinking carefully about each move you make, or perhaps in the associative stage, feeling comfortable with your performance but knowing there is room for improvement and still having to keep in mind things to avoid. Possibly you are at the autonomous stage of skill acquisition, having become an expert: your dancing makes use of various motor programs that you perform without thinking in response to sensory stimuli. If you are an experienced dancer, the skill memories you rely on reflect the coordination of several brain regions, including the basal ganglia, the cerebral cortex, and the cerebellum.

In short, there is more to social dancing than simply recalling and executing a fixed series of movements. It is an open skill in which the recent actions and reactions of your partner provide important feedback that you can use to guide your own actions. Keeping track of what has happened in the recent past is thus a key component of performing well on the dance floor. The ability to maintain and flexibly use memories of recent actions and their outcomes requires input from brain regions other than those we have focused on in the preceding discussion of skill learning and performance. We explore these kinds of memories and their neural substrates in [Chapter 9](#), on working memory.

Chapter 8 Review

Know Your Key Terms

associative stage

autonomous stage

basal ganglia

closed skill

cognitive skill

cognitive stage

constant practice

deep brain stimulation

expert

identical elements theory

implicit learning

knowledge of results

learning set formation

massed practice

mirror reading

mirror tracing

motor program

motor prosthesis

open skill

Parkinson's disease

perceptual-motor skill

power law of practice

rotary pursuit task

serial reaction time task

skill

skill decay

spaced practice

talent

transfer of training

transfer specificity

variable practice

Quiz Yourself

1. Surfing is an example of a(n) _____ skill because surfers have to adapt to variations in the qualities of each wave. ([p. 331](#))
2. Updating a website is an example of a(n) _____ skill because it requires using computer programming knowledge to solve a problem. ([p. 330](#))
3. Skiing is an example of a(n) _____ skill because it requires coordinating movements based on sensory information. ([p. 330](#))
4. Historically, researchers have questioned whether it is possible for animals other than humans to learn _____ skills. ([p. 333](#))
5. According to the _____, performance during learning improves rapidly at first, and then slows down. ([pp. 334–335](#))
6. People improve at the serial reaction time task without realizing that some sequences are being repeatedly presented or that their performance is improving, which suggests that their learning is _____. ([pp. 338–339](#))
7. If a baby is able to eat Grape Nuts using a spoon but shows no ability to eat them using a spork, then this may be a case of _____. ([p. 344](#))
8. A teenager who is learning to make macaroni and cheese for the first time is likely to be in the _____ stage of skill acquisition. ([p. 340](#))
9. Getting drunk will impair processing in your _____, which will make it harder to perform skills such as walking in a straight line. ([p. 359](#))
10. Cramming the night before an exam is an example of _____. ([p. 336](#))
11. Practicing the piano once a week for several months is an example of _____. ([p. 336](#))
12. Practicing a single dance routine that is synchronized to a specific piece of music is an

example of _____ practice. ([p. 337](#))

13. Practicing catching a baseball thrown at various heights, speeds, and distances is an example of _____ practice. ([p. 337](#))
14. The fact that professional athletes generally are not exceptional at all sports can be viewed as a case of _____. ([p. 344](#))
15. Thorndike proposed the _____ to explain why skills transfer best when they are used in situations that are highly similar to the one in which they were originally learned. ([p. 344](#))
16. Being able to type a text message without looking at the phone is an example of the _____ stage of skill acquisition. ([p. 340](#))
17. Neurons in the _____ change their firing patterns as rats learn to perform a perceptual-motor skill. ([p. 351](#))
18. In _____, extensive practicing of an instrument leads to loss of motor control. ([p. 356](#))
19. An age-related disorder involving a reduction in the number of dopaminergic neurons that affect basal ganglia activity is _____. ([p. 362](#))

Concept Check

1. A teenage boy wants to improve his dancing skills. What are some strategies he might try for learning these skills?
2. A graduate student who believes her pet tarantula is exceptionally bright wants to prove to the world that spiders can reason and solve problems. How might she convince others that she is correct?
3. Some researchers believe that the right kinds and amounts of practice can make anyone an expert. What sort of experimental evidence might convince these researchers that there is such a thing as talent?

4. According to Fitts's model of skill learning, individuals must go through an initial cognitive stage before they can master a skill. Does this imply that for a fish to learn to press a lever, it must first think about what is required to perform the task?
5. Neuroscience research has shown that regions in the somatosensory cortex and motor cortex expand in parallel with learning of perceptual-motor skills. Does this mean that practicing a skill causes regions of the cerebral cortex not involved in performing that skill to shrink?

Answers to Test Your Knowledge

Open and Closed Skills

1. Mostly open: balancing a ball on one's nose requires continuous feedback.
2. Mostly closed: swimming movements are predefined, and inputs are stable.
3. Mostly open if skilled: kissing requires continuous feedback.
4. Mostly open: fishing requires accurate prediction of changing inputs.
5. Depends on the fish and the insect. Mostly closed if the insect falls into the water; mostly open if the insect lives underwater.
6. Depends on the type of music. Mostly closed if classical; mostly open if jazz.
7. Mostly closed: dart-throwing movements are predefined, and inputs are stable.

Tasks That Test Skill Memory

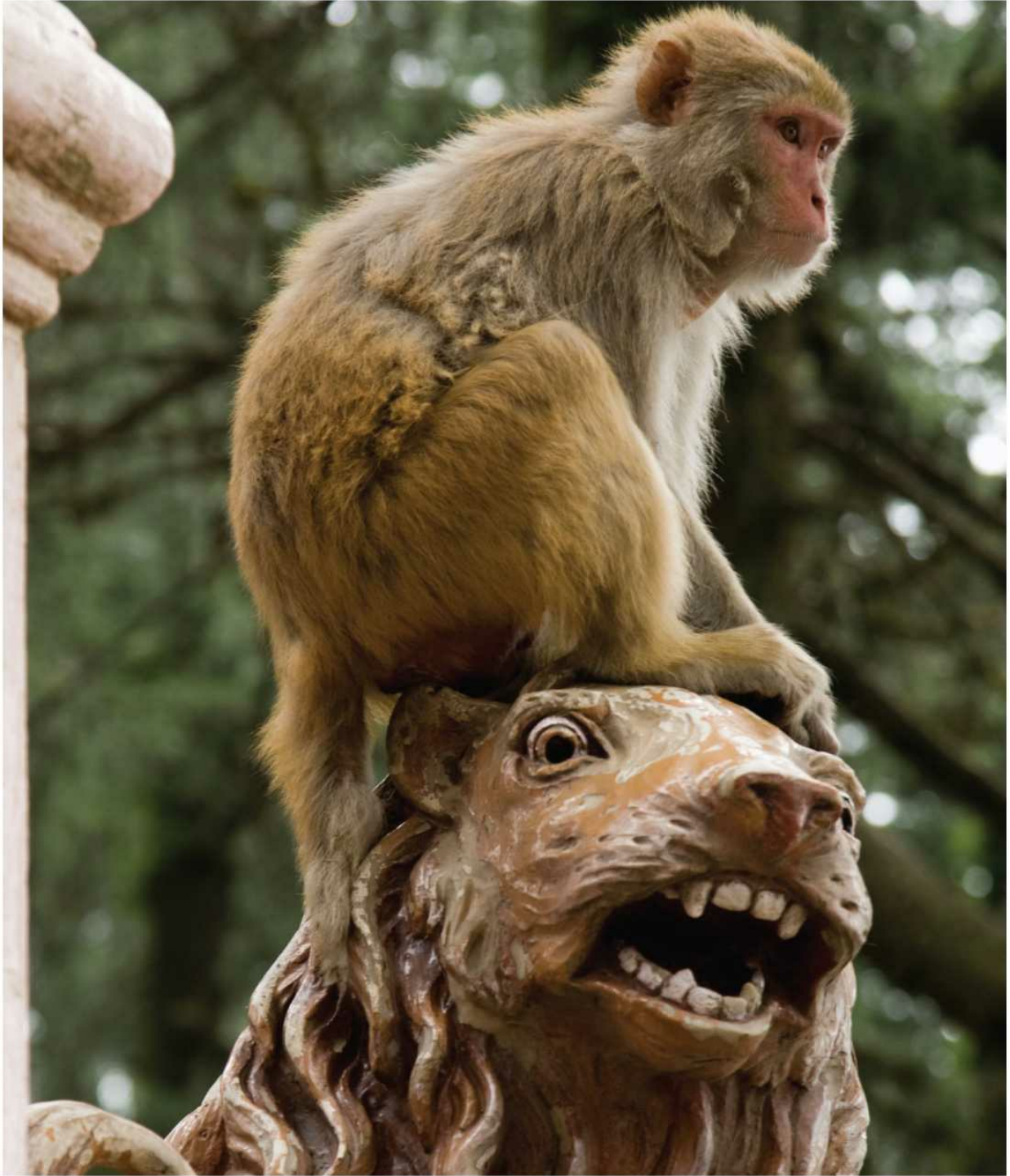
- The serial reaction time task is a perceptual-motor learning task that requires participants to press a key in response to a light as quickly as possible. It is used to test for unconscious (implicit) skill learning.
- The rotary pursuit task is a standard perceptual-motor skill learning task that measures how quickly individuals learn to coordinate their movements with a spinning disk. It has also been used to measure the role of genetics in determining how performance

changes with practice.

Which Cortical Regions Change During Skill Learning?

Behavioral studies (described in [Section 8.1](#)) show that chess experts visually scan the chessboard differently than do amateur chess players. Experiments with monkeys show that learning to distinguish tactile stimulation leads to changes in the somatosensory cortex, the cortical region involved in processing tactile inputs. In combination, these findings suggest that one cortical region where you might see differences between chess novices and grand masters is in the visual cortex, because it is a major source of the information that chess players use to select moves.

Chapter 9 Working Memory and Cognitive Control



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Behavioral Processes

[Transient Memories](#)

[Working Memory](#)

[Cognitive Control](#)

[*Learning and Memory in Everyday Life: Give Your Working Memory a Break*](#)

Brain Substrates

[Frontal-Lobe Anatomy and Consequences of Frontal-Lobe Damage](#)

[Studies of Frontal Brain Activity During Working-Memory Tasks](#)

[Mapping Executive Processing and Working Memory onto PFC Anatomy](#)

[Prefrontal Control of Long-Term Declarative Memory](#)

Clinical Perspectives

[The Prefrontal Cortex in Schizophrenia](#)

[Attention-Deficit/Hyperactivity Disorder](#)

IT IS TUESDAY AT 8:10 A.M., AND KAMILA, a high school senior, must rush if she is going to run several errands on the way to her first class—an 8:30 a.m. business class—and still arrive on time. She has only 20 minutes to get cash from the nearby bank machine, sign up for cheerleading tryouts, and drop off her biology homework, which is due by 10:00 a.m. Before heading out the door, Kamila grabs the various things she will need for the rest of the day, including her cheerleader pom-poms and iPad (for last-minute cramming for this afternoon’s quiz). The sign-up sheet for the tryouts is in the cafeteria, which is close to where her business class is being held. It is quicker, she figures, to go to the bank machine first, as that requires just a short detour from her homeroom. After signing up for cheerleading tryouts, she starts to walk to business class across campus when, *darn*, she realizes she forgot one of her errands. She doubles back quickly to the biology classroom, drops off her homework there, and then heads on to business class.

As she again walks toward her business class, Kamila remembers that today’s lecture has been moved from the usual third-floor location to the large auditorium in the basement, where the class will hear a guest speaker. She is so used to running up the stairs every morning that she has to struggle to remember that today she needs to bypass those stairs and take the elevator to the basement instead.

Slipping into the back of the auditorium, Kamila listens with half an ear to the speaker. Some of

the material he is presenting is new, but when he covers topics that are familiar from her own past reading, she switches her attention to the biology notes she's reviewing on her iPad (she's nervous about that upcoming quiz) and also to her iPhone lying discreetly in her lap; she is uploading photos from last night's awesome party to Instagram while checking to see how many "likes" her photos from last week's B'nai B'rith leadership retreat have been getting. This isn't the perfect situation for learning about business, studying biology, *or* keeping up with her busy social life, but by switching back and forth between the three activities, she manages to make some progress on all of them.

A day in the life of a high school senior is taxing indeed. To keep track of all her activities and commitments and deal efficiently with emergencies and last-minute changes, Kamila needs something like a mental notepad—a sort of imaginary easy-erase whiteboard for keeping track of information for short periods of time. In fact, that is a good description of her **working memory**, the active and temporary representation of information that is maintained for the short term in Kamila's mind to help her think and allow her to decide what to do next. As she attends to her various responsibilities during the day, Kamila's ability to control the flow of information into and out of her working memory is critical to the multitasking and planning she has to do to thrive in high school. **Cognitive control** is the manipulation and application of working memory for planning, task switching, attention paying, stimulus selection, and the inhibition of inappropriate reflexive behaviors. Thus, this chapter is about working memory itself as well as the broader range of cognitive control tasks in which working memory plays a crucial role.



Uncle Mark

How many different tasks is Kamila trying to do at once? What are the costs of multitasking?

working memory

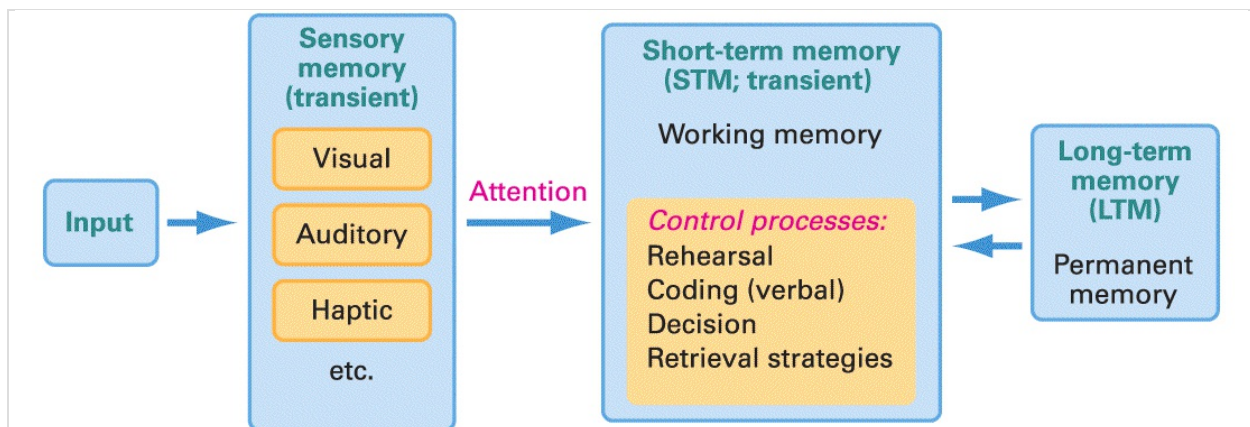
The active and temporary representation of information that is maintained for the short term, available for manipulation.

cognitive control

The manipulation and application of working memory for planning, task switching, attention paying, stimulus selection, and the inhibition of inappropriate reflexive behaviors. Also known as *executive control* or *executive function*.

9.1 Behavioral Processes

Most psychologists would agree that there are three main distinctions among different types of memory: *sensory memory*, where information automatically and rapidly decays; *short-term memory*, where information can be maintained as long as it is rehearsed or consciously attended to; and *long-term memory*, where memories can be retained for long periods, possibly permanently, without requiring ongoing maintenance or conscious attention. This view was laid out in an influential model by Richard Atkinson and Richard Shiffrin and is diagrammed in [Figure 9.1 \(Atkinson & Shiffrin, 1968\)](#). In this conceptualization of the stages of memory, **short-term memory (STM)** is the part of the memory system used as a temporary storage area and as the site of working memory (WM) operations.



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FIGURE 9.1 The Atkinson–Shiffrin model of memory Richard Atkinson and Richard Shiffrin’s model depicted incoming information as flowing first into sensory memory—shown as having distinct visual, auditory, and haptic (i.e., touch) and other sensory registers. Elements of sensory information that are attended to are then transitioned to short-term memory (STM). There they go through various control processes and in some cases are transferred into long-term memory (LTM).

Description

The information in the flow chart is as follows. Input leads to sensory memory (transient) which comprises visual, auditory, haptic memories (plus a few more which are not mentioned). The sensory memory unit perceived from attention leads to short-term memory (STM: transient) which is the working memory. The control processes of the working memory are listed as follows. Rehearsal, coding (verbal), decision, and retrieval strategies. Short term memory (STM) leads to long term memory (LTM) or permanent memory. LTM points back to STM.

short-term memory (STM)

A temporary memory that is maintained through active rehearsal.

The episodic and semantic memories discussed in [Chapter 7](#) are forms of **long-term memory (LTM)**, lasting for hours, days, or even years. (They are represented in [Figure 9.1](#) by the panel at the far right.) Skill memories, as discussed in [Chapter 8](#), can also be very long-lasting memories. In contrast, the memories that are the main focus of this chapter—short-term memories—are transient, existing briefly for seconds or minutes at most. These temporary memories are crucial for performing many high-level cognitive control functions, such as planning, organization, and task management.

long-term memory (LTM)

Permanent or near-permanent storage of memory that lasts beyond a period of conscious attention.

Transient Memories

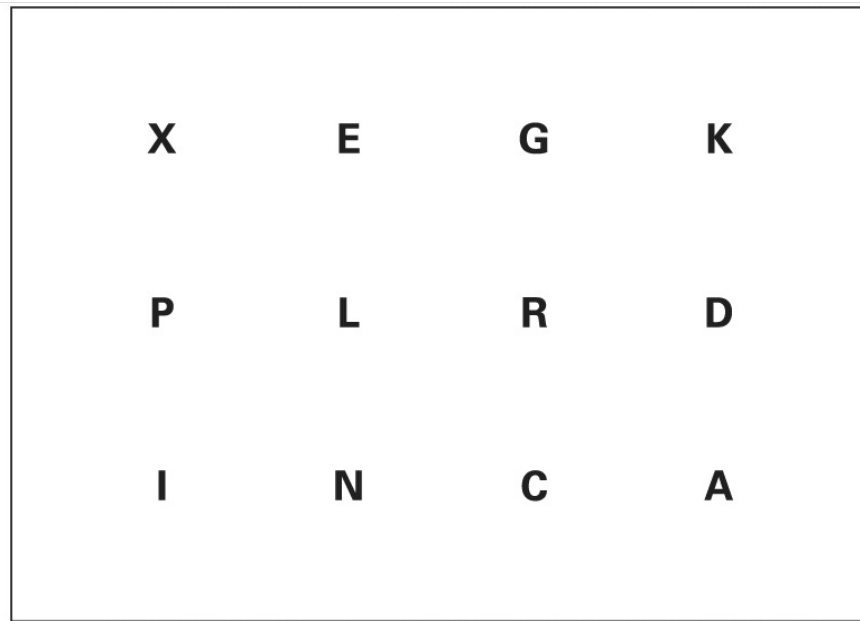
Transient memories are short-lasting mental representations, sometimes persisting for only a few seconds. The Atkinson–Shiffrin model describes two types of transient memory—sensory memory and short-term memory—and proposes that they correspond to the first two stages through which information from the world enters our consciousness and potentially becomes part of our long-term memory.

transient memory

Nonpermanent memory that lasts seconds or minutes; the Atkinson–Shiffrin model describes two types: sensory and short-term memory.

Sensory Memory

Sensory memories are brief, transient sensations of what you have just perceived when you have seen, heard, touched, smelled, or tasted something. Considerable research has been devoted to understanding how sensory memories are held in the mind so that they are accessible for further processing. Take a quick look at the table of letters in [Figure 9.2](#); just glance at it for a second—and no more. Now, without looking back at the figure, try to remember as many of the letters as you can. You probably recalled only four or five letters, or about 30–40% of the total array.



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FIGURE 9.2 The Sperling task These three rows of four letters each are similar to the array George Sperling used in his studies of visual sensory memory ([Sperling, 1960](#)). How many of the letters did you remember after glancing at them for a second?

sensory memory

Brief, transient sensations of what has just been perceived when someone sees, hears, or tastes something.

Based on this exercise, you might imagine that four or five items are the limit of your [visual sensory memory](#), the temporary storage in sensory memory for information perceived by your visual system. Perhaps, however, you felt as if your eyes saw more than four or five letters, but you just couldn't recall more of them. In a seminal 1960 paper, George Sperling conducted a study confirming that you probably did, very briefly, register more than just the few items you were able to recall ([Sperling, 1960](#)). Sperling presented his study participants with a three-by-four visual array, much like that shown in [Figure 9.2](#). He then played one of three tones after the array was removed. A high tone indicated that participants were to report the first row of letters, a medium tone corresponded to the middle row, and a low tone corresponded to the bottom row.

visual sensory memory

The initial temporary storage for information perceived by the visual system.

When this partial report procedure was used, participants were able to report about 75% of the letters overall. Note that this is about double the number of letters recalled when people are

simply asked to report as many letters as they can after the array is removed. What accounts for this doubled recall in Sperling's partial report procedure? Sperling interpreted it as meaning that people have a visual memory that persists for a very short time—less than a second—but that includes *all* the items recently seen.

If there is rapidly decaying sensory memory for visual information, you might imagine that there would also be an analogous rapidly decaying sensory memory for other sensory modalities, such as touch, smell, and hearing. Indeed, there have been studies showing similar phenomena with auditory memory ([Moray, Bates, & Barnett, 1965](#)). As illustrated in [Figure 9.1](#), there is a form of sensory memory for each sensory modality (auditory, visual, haptic—i.e., touch—etc.), which lasts very briefly and captures raw incoming sensory stimuli so that they can be processed and passed on to the short-term memory store, from which they may later be entered into long-term memory.

Short-Term Memory

Consider the common experience of looking up a phone number and then repeating it over and over to yourself as you prepare to press the buttons on your phone. The phone number has already been recognized and registered by sensory memory, but now it is the job of your short-term memory to maintain this information temporarily through active rehearsal. William James, whose work is presented in [Chapter 1](#), described short-term memory as being of limited capacity, effortlessly available, and fleeting in contrast to long-term memory, which holds permanent memories of the past, retrieved with effort, and which has potentially unlimited capacity ([James, 1890](#)). Short-term memory is, in James's view, the essence of our active conscious awareness. As summarized in [Table 9.1](#), short-term memory and long-term memory can be distinguished in several ways.

TABLE 9.1 Behavioral distinctions between short-term memory (STM) and long-term memory (LTM)

STM	LTM
Active contents of consciousness	Not currently in consciousness
Rapidly accessed	Accessed more slowly
Limited in capacity	Unlimited in capacity
Forgotten quickly	Forgotten more slowly

Your ability to retain information in short-term memory has certain limitations. First, as James noted, your memory is limited in capacity; a 10-digit phone number is a lot of information to keep in mind, even more so if you also have to remember a 4-digit extension. [Chapter 1](#) describes the classic studies of George Miller, who in the early 1950s suggested that the capacity of short-term memory is about 7 items—a number he described as “the magic number 7” because it recurred so frequently in studies of memory capacity ([Miller, 1956](#)). Actually, Miller argued that short-term memory capacities differ, usually numbering about 5 items but ranging from about 5 to 9 in most people (with the lower limits being more common).

Short-term memory is also limited to *what* you can pay attention to at the time. If you get distracted by something else, you are likely to forget all or some of the phone number as you walk across the room to get your phone: that is why you rehearse the number over and over in your head. By continuing to rehearse the number, you could potentially remember it indefinitely, as long as you do nothing else. Of course, there are plenty of things that could distract you and interrupt this rehearsal. If your roommate asks you a question—such as, “When is the chemistry exam?”—your rehearsal might be interrupted just long enough for you to forget some or all of the phone number. If you do forget it, you have to look it up again.

The capacity of short-term memory is also determined by how we encode the information we try to retain. As noted previously, most people can keep only about half a dozen random numbers in short-term memory. Given this limited capacity for short-term memory, you might think it impossible to keep the following 11 digits in mind without memorizing them: 91117761492. Actually, it is quite easy, because you really only need to remember three historic dates: the month–day reference to the attack on the World Trade Center (9/11), the signing of the U.S. Declaration of Independence (1776), and Columbus’s first visit to America (1492). Miller argued that the limit to short-term memory is not an absolute amount of information (or number of digits) but rather a limit to the number of unique concepts or links to long-term memory that can be held active. Miller argued that recoding of information is key to optimizing the amount of information that can be maintained in short-term memory.



Left: Sean Adair/Reuters/Newscom; top right: Todd Taulman Photography/Shutterstock; bottom right: Sarin Images/GRANGER — All rights reserved.

What 11 digits do these three images convey?

Description

Photo 1: A photo shows footage documenting planes hitting the World Trade Center on September 11, 2001. The towers are engulfed in flames, and thick smoke emanates from them.

Photo 2: An image of The Declaration of Independence.

Photo 3: A portrait shows a group of eighteenth-century soldiers by a waterfront, with swords and flags.

It is no accident that 10-digit American phone numbers are broken into three chunks of 3, 3, and 4 digits, respectively. Chunking is an essential tool for controlling our short-term memory and using it effectively to temporarily store as much information as possible. Note that in the case of storing several historically significant dates to increase capacity to 11 digits, you must utilize your long-term memory to retrieve the dates from your knowledge of American history. As you will read later, such interactions between short-term memory and long-term memory are critically important.



*“These drugs will affect your short-term memory
so you better pay me now.”*

Paul Taylor/CartoonStock.com

Working Memory

Rehearsal, then, is an important part of how we keep information active and accessible within short-term memory. As you walk across the room from your computer—where you found the phone number—to the phone, you rehearse the number to keep from forgetting it. Your goal is not necessarily to store the phone number in your long-term memory (although doing so might be useful for future reference); rather, your immediate aim is to remember the number just long enough to get to your phone and make the call.

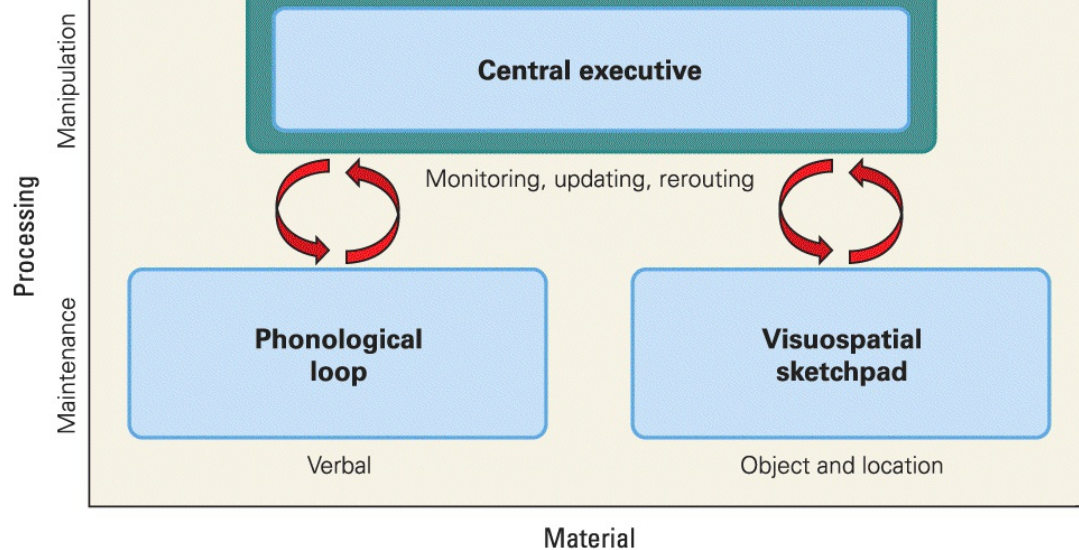
Short-term memory used in this way serves as a buffer, or temporary holding station, maintaining information for a brief period before it is manipulated or otherwise used to affect behavior. It is when short-term memory is subsequently put to use to perform a task of some kind that it is referred to as working memory. Thus, working memory involves the temporary retention of information just experienced or just retrieved from long-term memory for the purpose of making decisions, retrieving other information, or performing other manipulations leading to goal-directed behavior. These processes will be described in more detail next.

It is important to realize that short-term memory and working memory are not two different types of memory. Rather, *short-term memory* refers to the broad class of transient memories that receive our attention; and working memory is one of the many ways in which short-term memory is used as a cognitive tool. Thus, working memory can be thought of as a special case of short-term memory. We use short-term memory as a working memory buffer when we briefly hold some ideas “in mind” and deploy them to help direct behaviors or help determine other cognitive processes, such as the retrieval of information from long-term memory. The distinction between how we use the terms *short-term memory* and *working memory* is subtle but important. Although there are many aspects to short-term memory, and many ways in which short-term memory is important to cognition and behavior, most of this chapter focuses on the role that short-term memory plays when it is employed as a working-memory buffer.

Baddeley’s Working-Memory Model

Alan Baddeley, an English psychologist, proposed an influential model of working memory, illustrated in [Figure 9.3](#) ([Baddeley & Hitch, 1974](#)). It can be viewed as an alternate (and more detailed) version of what goes on inside the STM/working-memory box in [Figure 9.1](#).

Baddeley’s model includes two independent short-term memory buffers: the visuospatial sketchpad and the phonological loop. The [visuospatial sketchpad](#) holds visual and spatial images for manipulation. The [phonological loop](#) does the same for auditory memories, maintaining them by means of internal (subvocal) speech rehearsal (much like a “loop” of a tape recording that goes around and around, playing the same song over and over). A key feature of Baddeley’s theory is that visuospatial information and verbal–phonological information are stored separately as distinct parts of working memory.



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FIGURE 9.3 Baddeley's working-memory model This model consists of two material-specific temporary stores for short-term maintenance of information—a visuospatial sketchpad for object and location information and a phonological loop for verbal material—that are controlled by a central executive. It also distinguishes between two general processes of working memory: maintenance and manipulation. Baddeley's model can be viewed as depicting what goes on inside the STM box in [Figure 9.1](#).

Description

The information in the flowchart is as follows. A central executive on top responsible for monitoring, updating, and rerouting is linked by two directional arrows to a phonological loop (verbal) and a visuospatial sketchpad (object and location). The central executive is responsible for the manipulation and processing of verbal information and information related to object and location. The phonological loop and the visuospatial sketchpad are responsible for the maintenance of verbal information and information related to object and location.

visuospatial sketchpad

The component of Baddeley's model of working memory that holds visual and spatial images for manipulation.

phonological loop

The component of Baddeley's model of working memory that maintains auditory memories by internal (subvocal) speech rehearsal.

A third component of Baddeley's model is the [central executive](#), which monitors and manipulates both of these working-memory buffers, providing cognitive control of working memory. The central executive's manipulations include adding to and deleting from the items in the buffers, selecting among the items in order to guide behavior, retrieving information from long-term memory, and transferring information from the visuospatial sketchpad and

phonological loop to long-term memory. In these ways, the central executive manages the work that takes place in short-term memory and the traffic of information back and forth between short-term and long-term memory.

central executive

The component of Baddeley's model of working memory that monitors and manipulates the two working-memory buffers.

[Figure 9.3](#) highlights two important distinctions made by Baddeley's model. First, it distinguishes between two general processes of working memory: manipulation (which depends on the central executive) and maintenance (which requires only rehearsal of information in the two memory buffers). Second, it identifies the memory buffers as being material specific: one stores verbal material, and the other stores object and location material. We will proceed now with a discussion of the two memory buffers: the phonological loop and the visuospatial sketchpad. Later, in [Section 9.2](#), we will discuss recent data from neuroscience suggesting how both the material-specific and process-specific dichotomies in Baddeley's model of working memory have guided research on the brain mechanisms of working memory and cognitive control.

The idea of material-specific buffers in short-term memory received support from a series of studies testing selective interference. This research posited that if two tasks are using the same processing mechanism, they should interfere with each other if performed at the same time. Indeed, a wide range of studies show that verbal tasks interfere with verbal short-term memory but not with visual short-term memory and that visual tasks interfere with visual short-term memory but not with verbal short-term memory. This finding lends support to the notion that working memory contains independent, material-specific systems for verbal and visual memories. The following paragraphs review some of these data.

The Phonological Loop

Read this list of seven numbers: 5 6 2 8 1 7 3. Now look away for 5 seconds and then repeat the list out loud. How did you solve the problem of remembering the numbers in this digit-span test? Most likely, you rehearsed them silently in your mind during the interval. In fact, if you did not rehearse the numbers, you probably wouldn't have been able to remember them. Without rehearsal, most people retain about 2 seconds' worth of information in their phonological memory, which gives most people enough time to remember about 5 to 7 items—again, Miller's

magic number 7. Because of this time limit, people with slow rates of speech but normal intelligence do worse on short-term verbal memory tasks than do people of normal intelligence who speak at a normal rate ([Raine, Hulme, Chadderton, & Bailey, 1991](#)). A person's internal speech proceeds at about the same rate as the person's speech spoken aloud. This internal, unspoken speech used during rehearsal is vital to the phonological loop and verbal working memory.

In fact, if this internal rehearsal is disrupted or eliminated, phonological storage cannot occur. For instance, if you were to say out loud, “Good morning, good morning ...” during the delay period while you were trying to remember the list of numbers in the digit-span test, your ability to internally rehearse would be greatly disrupted, impairing your ability to recall the seven numbers. On the other hand, tapping your fingers (a motor task) does not interfere with internal vocal rehearsal because it requires different modalities of processing—that is, speech versus motor.

Additional insights into the processes for internal rehearsal in short-term memory come from studies in which people are asked to remember lists of words. For example, which list do you think would be easier to remember?

List 1: bat, hit, top, cat, door

List 2: university, expedition, conversation, destination, auditorium

Most people would say the first is easier. As the length of the words increases, the number of words you can remember declines; this is known as the [word-length effect](#). Short, one-syllable words like “bat” and “hit” are easier to rehearse in working memory than longer, multisyllable words like “university” and “auditorium.” Longer words take longer to rehearse ([Baddeley, Thomson, & Buchanan, 1975](#)).

word-length effect

The tendency for a person to remember fewer words from a list as the length of the words increases.

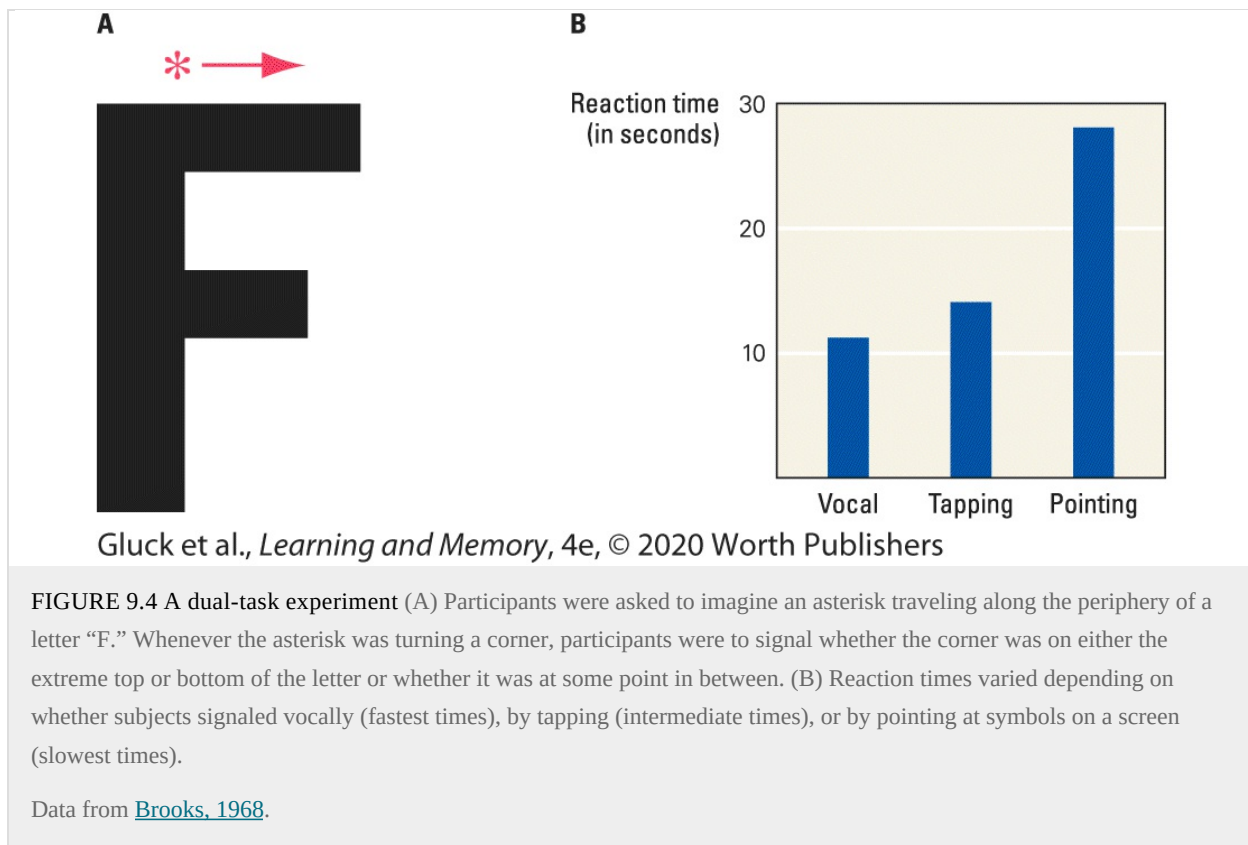
The Visuospatial Sketchpad

The visuospatial sketchpad in Baddeley's model of working memory (see [Figure 9.3](#)) is a mental

workspace for storing and manipulating visual and spatial information. Here is an example of it in use: without writing anything down, picture a four-by-four grid (16 squares) in your mind and imagine a “1” in the square that is the second column of the second row. Then place a 2 to the right of that. Next, in the square above the 2, put a 3, and to the right of that put a 4. Below the 4, put a 5 and below that, a 6, and then to the left of that, a 7. Now, what number is just above the 7? To correctly answer this question (2), you had to use your visuospatial sketchpad.

Just as the phonological loop has a 2-second time limit, the visuospatial sketchpad also has a limited capacity. The two capacities, however, are independent: filling up one does not much affect the capacity of the other. Dual-task experiments, in which subjects are asked to maintain information in the visuospatial sketchpad while simultaneously carrying out a secondary task using the other modality—such as retaining an auditory list of words in the phonological loop—provide evidence for the independence of these two memory buffers.

For example, Lee Brooks used a dual-task paradigm in which people were shown a block-capital letter “F” and were then asked to visualize this letter (from memory) and imagine an asterisk traveling around the edge of it ([Figure 9.4A](#); [Brooks, 1968](#)). When the imaginary asterisk reached a corner, it turned left or right to continue following the outline of the letter F. At each such turning point, the people were asked to indicate whether the asterisk was at a corner at either the extreme top or bottom of the F (e.g., the point at the F’s upper right) or whether it was at one of the in-between corners. The crucial manipulation was that the participants were divided into three groups, and each group was assigned a different way of signaling. The *vocal group* signaled an answer to each question with a verbal “yes” or “no,” the *tapping group* signaled with one tap for yes and two taps for no, and the *pointing group* pointed to a visual array of Y’s and N’s on a screen. Of the three groups, the pointing group performed most slowly, suggesting that the visuospatial demands of pointing at the appropriate symbol interfered with the visuospatial memory task ([Figure 9.4B](#)).



Description

Section a: the letter F is shown in a bigger font. An asterisk with a rightward arrow next to it, is shown on top of the upper boundary of the letter.

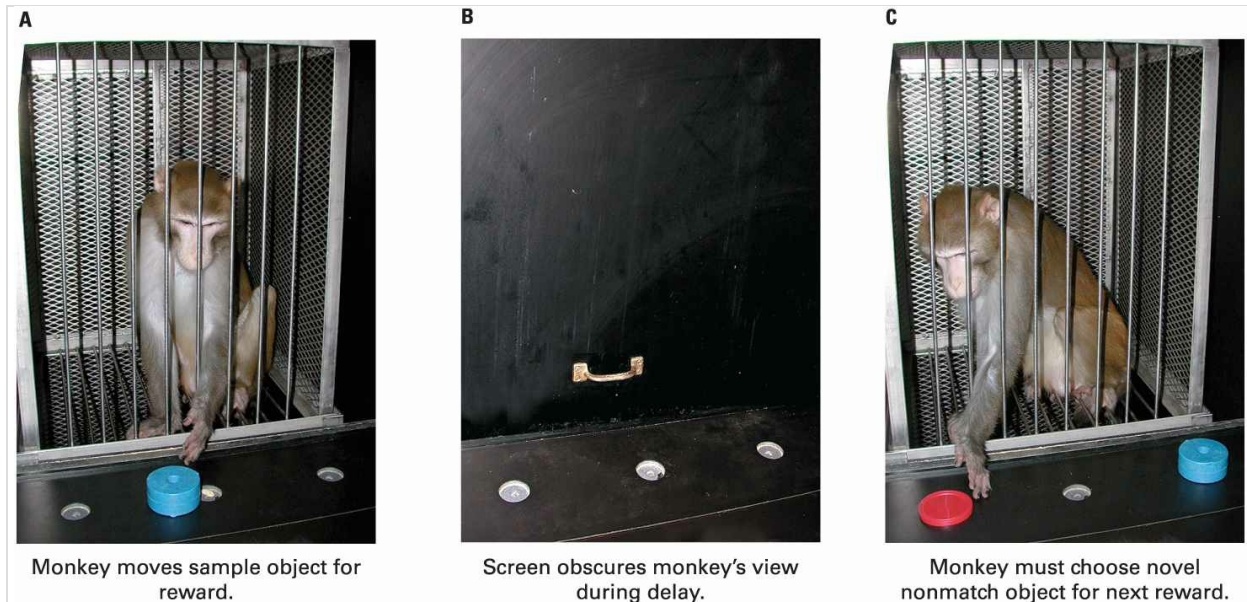
Section b: A bar graph shows the reaction time of vocal, tapping, and pointing signals compared to time in seconds. The readings on the vertical axis range from 0 to 30 in increments of 10. The reaction times for different signals are as follows. Vocal-12 seconds, tapping-14, pointing-28 seconds.

All the above data is approximate.

Because visual memory can be easily studied in a wide range of species, it has become the modality of choice for many carefully controlled laboratory experiments on working memory in animals. For instance, in an early study of spatial working memory, Carlyle Jacobsen trained monkeys on a delayed spatial-response task ([Jacobsen, 1936](#)). Each monkey watched food being placed in one of two bins (left or right). Next, an opaque screen came down and blocked the monkey’s view of the bins for several seconds or minutes. The screen was then lifted to reveal that the bins were now covered by lids hiding the food. To be deemed correct, the monkey first had to remember in which bin the food had been stored and then displace the lid of just that bin

to retrieve the reward.

The **delayed nonmatch-to-sample (DNMS) task** is another test of visual memory. Each trial involves remembering some object seen at the trial's start. **Figure 9.5A** shows Pygmalion, a rhesus monkey in Mortimer Mishkin's laboratory at the National Institute of Mental Health. He is shown a "sample" object, a blue ring, under which he finds a food reward, such as a peanut or a banana pellet. Afterward, an opaque black screen obscures Pygmalion's view (**Figure 9.5B**) for a delay period ranging from seconds to minutes, depending on the experiment design. During this delay period, the experimenters introduce a new object, a red disk. When the screen is raised, Pygmalion sees both objects, one on the right and the other on the left. As shown in **Figure 9.5C**, Pygmalion has learned that a reward will now be found under the red disk because this is the novel object, a "nonmatch" to the sample object he saw previously. Training on this delayed nonmatch-to-sample task continues for several trials, each of which involves two objects not used in previous trials. Thus, the next trial might involve a yellow box as the sample and a green disk as the novel object. Over many such trials, the correct answer is sometimes on the left and sometimes on the right so that spatial location will not be a useful cue.



Courtesy of David Yu, Mortimer Mishkin, and Janita Turchi, Laboratory of Neuropsychology, NIMH/NIH/DHHS.

FIGURE 9.5 Delayed nonmatch-to-sample task (A) A monkey is shown a sample object (here, a blue ring), and finds a reward under it. (B) During the delay period, an opaque black screen blocks the monkey's view of any test objects. (C) The monkey is shown two objects, the blue ring from before (the sample) and a new object, a red disk. Through training, the monkey has learned that a food reward will be hidden under whichever object is the new one, the nonmatch to the original sample. The monkey chooses the nonmatch.

Description

Photo A: The monkey stretches its hands through the bars of the cage toward a blue ring. The text reads, monkey moves sample object for reward.

Photo B: An opaque shutter closes the cage. The text reads, screen obscures monkey's view during the delay.

Photo C: The monkey stretches its hand toward a red ring, which is placed in addition to the blue ring shown earlier. The text reads, monkey must choose novel nonmatch object for next reward.

delayed nonmatch-to-sample (DNMS) task

A test of visual memory in which a subject must indicate which of two objects is not the same as the sample object that was recently seen.

Because each trial uses a new set of objects, a monkey must learn to remember which unique sample he saw previously and hold this memory in his visuospatial memory buffer until presented with the choice of that previous sample and the new novel object. With short delays, the DNMS task can be used to assess working memory; with longer delays, however, the same task has also been used to study long-term memory.

Is Working Memory a Place or a State?

Working memory is often conceptualized as the mind's active workspace—something akin to the work surface of your desk, where you bring files that have been stored elsewhere, physically moving them to where you can work with them all in one place. This idea of two different places, a long-term memory (LTM) and an active working memory, originated in the pre-computer days, when LTM was described using the metaphor of file cabinets filled with manila folders that had to be retrieved from storage in order to be used.

This metaphor of a place for working memory was further ingrained when computer systems came into use. Computer hardware distinguishes between a large hard drive and a smaller set of random-access memory (RAM) chips. The RAM chips in a computer define how many programs and how much data you can work with at any one time (like the limits on working memory), while the hard drive, with far more storage capacity, defines the total limits on memory storage for all your files and information, even those you have not looked at or thought about in a long time (such as emails to your high school boyfriend or last year's chemistry homework).

The idea of working memory and LTM as being two concrete, physical places in the mind dominated the first several decades of research on working memory. The Atkinson–Shiffrin model in [Figure 9.1](#), where information is diagrammed as flowing from STM/WM to LTM and back, seems naturally to suggest that memories move back and forth between these places. There is, however, another way of thinking about these transitions that has come to the forefront of working-memory research in recent years. “Working memory” might not, in fact, describe a separate *place* for memories to be moved; rather, it might describe an active *state* for memories otherwise resident in LTM but not accessible to conscious reflection and manipulation until they are activated.

Consider an art museum that has a vast collection of paintings, far more than can be viewed at any one time. One way to focus on a small subset of the paintings might be to bring them out, a few at a time, to a special viewing workroom. This is akin to the place model of working memory. If the workroom is very small (far smaller than the museum), you might be able to display only a few of the paintings at a time. Alternatively, you could leave all the paintings where they are stored on the walls of the vast halls of the museum and place several high-intensity lights to shine on the specific paintings you want to observe. The paintings stay where they always are, but the state of the paintings changes: they go from being obscure and unseen to being illuminated and visible.

As appealing and as useful as it has been for researchers in psychology to think of working memory as a place in the mind or brain, a growing body of research has shifted the field toward thinking about working memory as being more like an illuminated state and less like a physical place. Does this mean the Baddeley model is wrong? Not entirely. However, the state model of working memory does suggest that the central executive and phonological and visuospatial sketchpads of [Figure 9.3](#) might better be thought of as states of activity and processing rather than as physically distinct places within memory. The phonological loop and the visuospatial sketchpad could then be reinterpreted as the active states of phonological and visuospatial memories rather than as physically distinct places or components of memory.

[Nelson Cowan \(1995\)](#) has described a model of short-term memory in which several chunks of information in LTM can be activated as the focus of current attention. Recently activated items in LTM that are no longer the focus of attention can, for some limited period of time, remain

partially activated and thus can be more easily brought back into focal attention. This description would imply that STM is characterized by at least two different types of activated states, one directly accessible to attention and the other somewhat removed from the current attentional focus but easily brought back into focus. A number of variants on this state-based model of short-term and working memory have been proposed ([Awh & Jonides, 2001](#); [Luck & Vogel, 2013](#); [Oberauer, 2002, 2013](#)).

Place models of memory are known as “multi-store” models of memory because they imply the existence of two or more different places for memories to be stored; accordingly, state-based models of memory have been referred to as “unitary-store” models of memory because they imply that there is only one place for memory, although the memories can be in various states.

A key question for all researchers in this subject area has been how much information can be stored in STM ([Jonides et al., 2008](#)). As reviewed earlier, Miller argued that STM has a capacity of “seven plus or minus two” ([Miller, 1956](#)). Place models of memory view these capacity limits as being governed by the rate at which information is rehearsed, forgotten, and transferred from one store to another ([Baddeley, 1986](#)). In contrast, state-based models of STM view capacity limits as arising from the bandwidth of attention that can be focused on activated areas of LTM ([D’Esposito & Postle, 2015](#)).

Overall, state-based models of working memory are able to account for much of the same data as the place-based models of working memory. The principal reason they have gained prominence in recent years is that they are better able to account for new brain-imaging data from cognitive neuroscience studies of the brain substrates of working memory. We will discuss these studies in more detail later in this chapter, in [Section 9.2](#).

TEST YOUR KNOWLEDGE

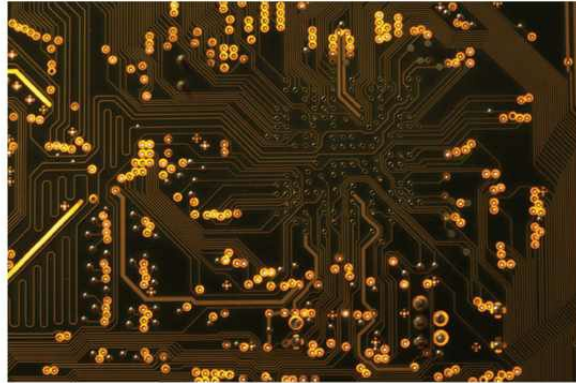
Models of Working Memory

Identify each of these images as analogous to either a place model of working memory or a state-based model. ([Answers appear at the end of the chapter.](#))

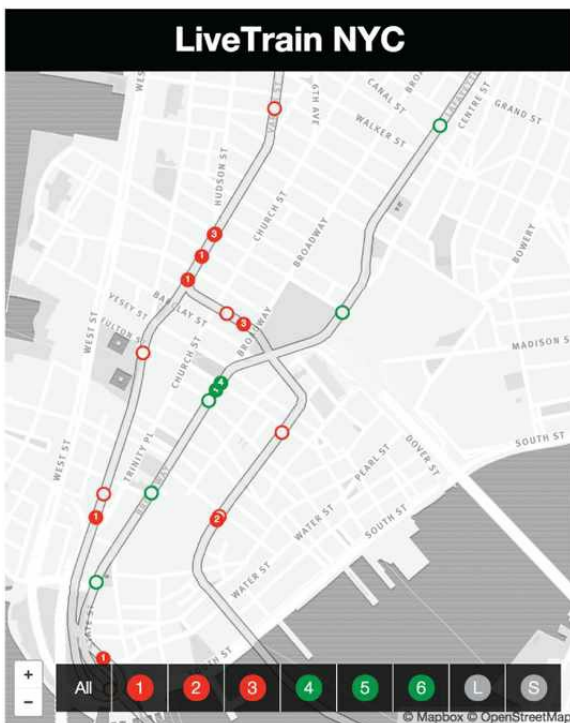
A



B



C



D



A: thodonal88/Shutterstock; B: NV77/Shutterstock; C: Courtesy of LiveTrain.nyc; D: Marc Rasmus/imageBROKER/Alamy Stock Photo

Description

Photo A shows a laptop on a table with papers scattered all over. In the background, there is a shelf containing files.

Photo B shows a microcircuit board.

Photo C shows a map displaying the live status of a train in N Y C, with red, and green numbered spots on it. A tab at the bottom shows these options: All, followed by three red circles numbered from 1 through 3 and three green circles numbered from 4 through 6. Two gray circles are labeled L and S.

Photo D shows different newspaper extracts posted on a wooden surface.

Cognitive Control

Most of the tasks described in the preceding section focus on the ability of the person or animal to maintain some word, digit, object, sound, or location in working memory during a delay period. But there is much more to working memory than just the maintenance of phonological or visuospatial memories; there is the far more complex and involved process of *manipulating* working memory. This is *cognitive control*, also known as *executive control* or *executive function*, the manipulation of working memory that allows for the exercise of various aspects of higher-order cognition, including reasoning, task flexibility, problem solving, and planning.

We saw earlier, for example, that Kamila has to keep several of her day's goals in mind: get cash, prepare for cheerleading, study for biology, and listen to a business lecture. Balancing these multiple goals requires her to switch her attention back and forth between activities as the situation requires. Her working memory is constantly being updated and reorganized to accomplish different and competing tasks. New tasks are constantly added. All of these tasks require the cognitive-control function of her working memory's central executive.

Of the three components of Baddeley's model, the central executive is the most important, the most complex, and the least well understood. What is common to all the functions of the central executive is that they involve the *manipulation* of information in short-term memory, including adding or removing items, reordering items, and using working memory to guide other behaviors. Through this manipulation of information held in short-term memory, the central executive goes beyond simple rehearsal to become, in effect, the *working* component of working memory.

Researchers have found evidence of cognitive control in many behaviors, including, but not limited to, (1) controlled updating of short-term memory buffers, (2) goal setting and planning, (3) task switching, and (4) stimulus attention and response inhibition. We will discuss each of these in turn in the following pages; they are listed, along with tasks used to study them, in [Table 9.2](#). This list of behaviors may seem, at first glance, like a random selection of activities, but they all depend on the controlled manipulation of working memory. The list is by no means

exhaustive—there are many other behaviors that involve working-memory manipulation—but these four examples serve to illustrate the breadth and diversity of mental activities that involve working memory and cognitive control.

TABLE 9.2 Cognitive control through the manipulation of working memory

Behaviors	Tasks used to explore these behaviors
Controlled updating of short-term memory	<i>N</i> -back task, self-ordered search
Goal setting and planning	Tower of Hanoi
Task switching	Wisconsin Card Sorting Test
Stimulus attention and response inhibition	Stroop task

Controlled Updating of Short-Term Memory Buffers

The central executive component of working memory functions much like a manager at a large corporation who is responsible for assigning specific people to certain jobs at particular times. On Monday, he might tell Mike to work the front desk and Stephanie to work on the sales floor. Come Tuesday, however, he might fire Mike, promote Stephanie to the front desk, and then hire Kristy to work on the sales floor. In an analogous fashion, the central executive in Baddeley's model is responsible for updating working memory by receiving and evaluating sensory information, moving items into and retrieving them from long-term memory, and deciding which memories are needed for which tasks.

To study the controlled updating of working memory, researchers often use what is called a *2-back test*. In a 2-back test, a participant is read a seemingly random list of items, usually numbers. A certain item—let's say the number 7—is designated as the “target.” Whenever the target number 7 is read, the participant is to respond with the number that was read *two numbers previously* (hence the name *2-back*). Sound tough? Try it. If the numbers read aloud are 4 8 3 7 8 2 5 6 7 8 0 2 4 6 7 3 9 ... , what would the correct responses be? (Answers: “8” to the first 7, “5” to the second 7, and “4” to the third 7.)

To succeed at this task, the participant must constantly keep track of the last two numbers that were read: the 1-back and 2-back numbers. As each new number is read, a new 1-back number must be stored and the old 1-back number must be shifted to the 2-back slot, replacing the

previous 2-back number in working memory. In addition, each number must be checked as it is read to see if it is the target number. If it is the target number, the participant has to respond with the 2-back number; if it is not the target number, the participant says nothing. Not easy!

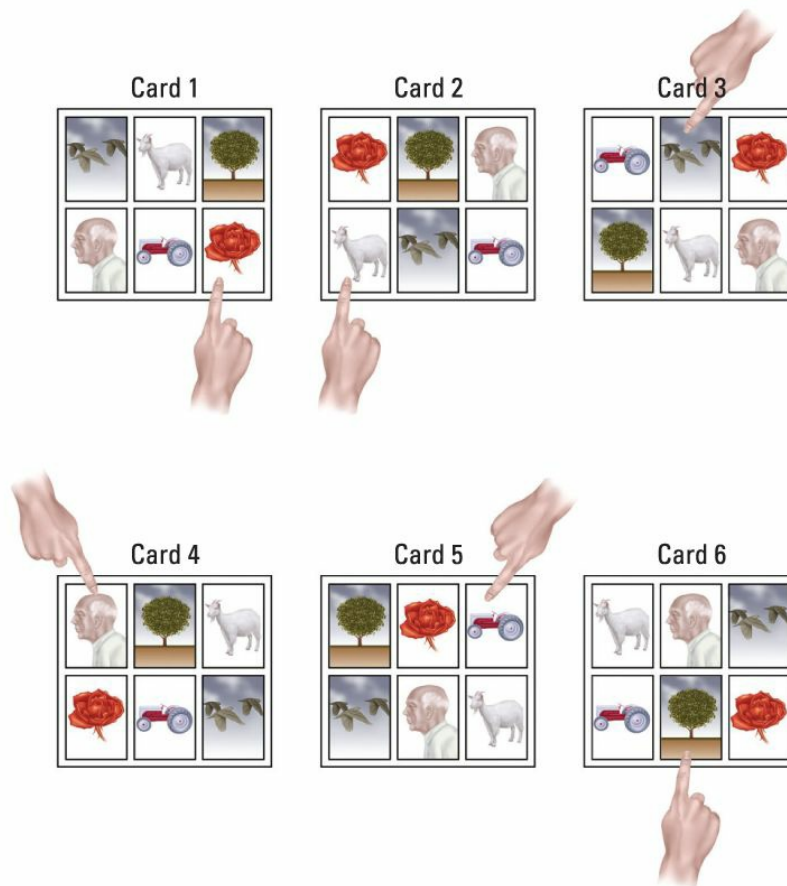
Performing the 2-back task requires active maintenance of many kinds of items in working memory. First are the target number and the rules for performing the task, both of which stay constant throughout the experiment. Second, the last two numbers that were read must always be remembered in case the next number is the target number. These two items change in identity, priority, or both with each new number that is read and must be regularly updated in working memory.

What might happen to your performance on this type of task if you were asked to repeat the 3-back or 4-back number instead of the 2-back number? Although the 2-back task is the most commonly used variation, this class of tasks is generally called *N*-back because *N* can be any number. The larger *N* is, the greater the challenge. The *N*-back task taps into many aspects of the central executive's manipulation of working memory, including online storage of recent information, selective attention, remembering task demands, and updating and reorganizing stored items. For this reason, it is considered an excellent tool for assessing the functioning of working memory's central executive. We will discuss experimental studies of the *N*-back task in [Section 9.2](#).

A more common situation faced by your own central executive functioning is the need to keep track of the various everyday tasks you need to perform: What have you done already? What remains to be accomplished? For instance, if you have lost your eyeglasses *somewhere* in your home, you might search every room for them. While it may not matter which rooms you search first, you do want to keep track of the rooms so as not to waste time searching the same room twice. Self-ordered tasks that ask people to keep track of their previous responses (analogous to keeping track of the rooms they already searched) are another tool that can be used to assess the central executive's manipulation of working memory.

Michael Petrides, at McGill University in Canada, developed self-ordered memory tasks in studying the behavioral and neural bases of working memory ([Petrides, 2000](#); [Petrides & Milner, 1982](#)). In the human version of this task, people are shown a stack of cards, each containing the

same set of items but in a different order. In the example shown in [Figure 9.6](#), there are six items on each card. In card 1, the drawing of a rose (to which the subject is pointing) appears in the lower-right corner, whereas in card 2 the rose is in the upper-left corner, and in card 3 it is in the upper-right corner.



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FIGURE 9.6 A self-ordered memory task for humans Sample cards from a self-ordered search task for humans ([Petrides & Milner, 1982](#)). Participants are presented with a stack of cards, each containing all the items in the target set but in different random order. Participants must point to a different item on each card without repeating any of the items.

Information from [Petrides, 2000](#).

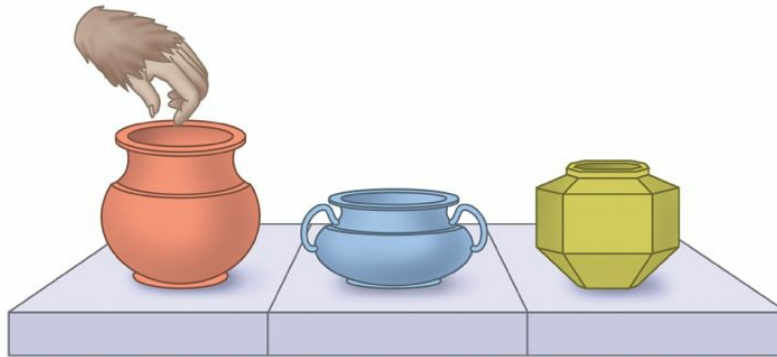
Description

The illustration shows six cards, numbered from 1 through 6. Each card displays the same six objects but arranged in a different order. A finger points toward a different object in each card.

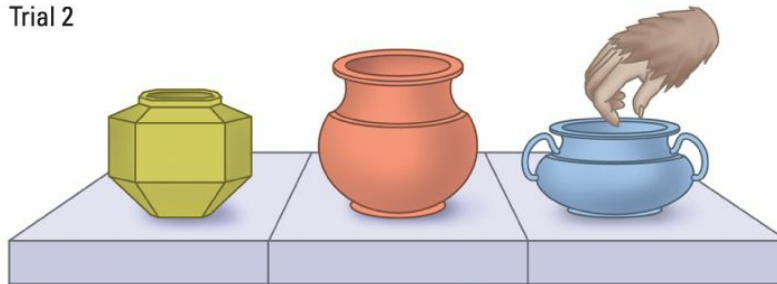
The experiment proceeds as follows: On trial 1, a participant is shown the first card and is asked to choose any of the six items on it. The participant in [Figure 9.6](#) has chosen the rose. This card is then flipped over. Next, on trial 2, the participant is shown the second card (with the same six items in a different order) and is asked to choose any of the five items not yet selected. In [Figure 9.6](#), the participant has chosen the goat. This second card is then flipped over. Then the participant is shown the third card and must pick any of the four remaining items that were not chosen on the previous two cards—that is, any image except the rose or the goat. This self-ordered task continues until the participant has pointed to all six different items without repeating any. Note that this task requires the participants to monitor (keep track of) the information being held in short-term memory and decide what to keep there, given the current task.

This task is appealing to researchers who want to understand working memory in both human and nonhuman primates because it can also be studied in monkeys, as shown in [Figure 9.7](#) ([Petrides & Milner, 1982](#)). On the first trial, a monkey sees a row of three nonmatching containers, each of which holds a reward, and selects the reward from one of them. Following this step, an opaque screen is placed between the monkey and the containers for 10 seconds, and the containers are shuffled so that on the second trial the monkey sees the same containers in a new order. Now the monkey must choose one of the other containers in order to get a reward. Like the human self-ordered task described above, this task requires the monkey to remember the items chosen previously. On the third trial, the monkey has to choose again, with only one remaining container still baited with a reward. Because this kind of working-memory task can be performed by both monkeys and humans, it is useful for comparative studies of the neural substrates of working memory, as will be described in [Section 9.2](#).

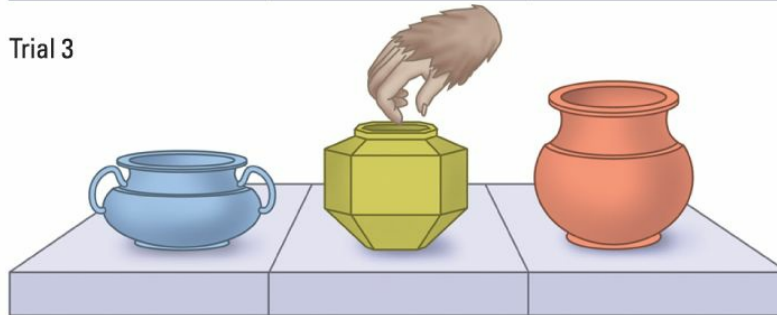
Trial 1



Trial 2



Trial 3



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FIGURE 9.7 A self-ordered memory task for monkeys The monkey sees three distinct containers and selects a reward from one of them. Their order is shuffled on each trial, and the monkey must remember which containers have had rewards removed so that on subsequent trials the monkey does not pick a previously chosen (and hence empty) container.

Description

The illustration shows three differently shaped containers arranged on a platform. They are arranged in a different order during trials 1, 2, and 3. A hand of a person is shown near different containers during each trial.

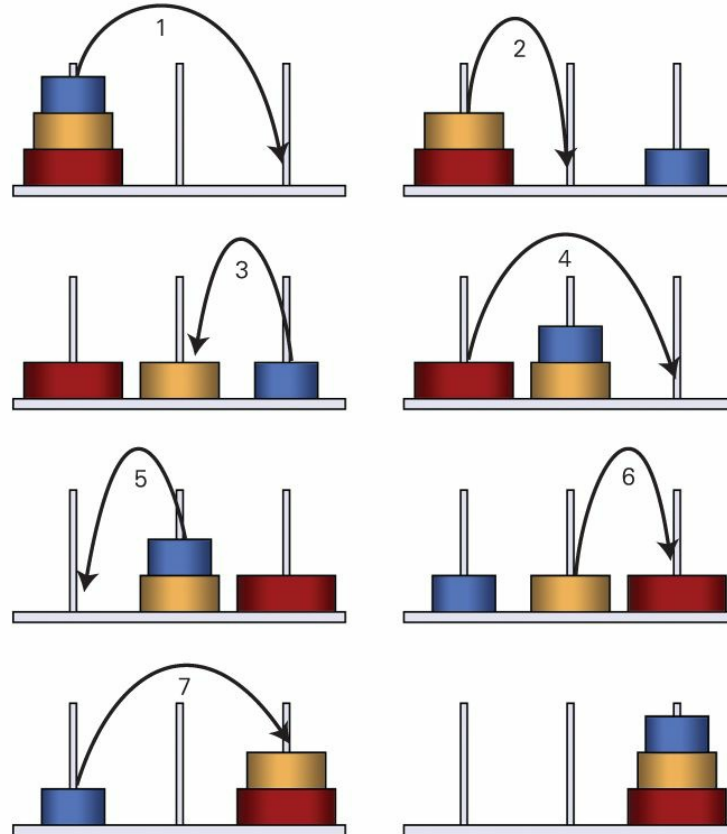
Goal Setting and Planning

As Kamila prepared for the school day ahead of her, she had to be aware of her immediate goals (getting to business class on time in the morning) as well as her goals for later that afternoon (cheerleading tryouts). To make sure she could participate in the tryouts and get to them on time

after her afternoon class, she had to (1) search through her closet to find her cheerleading pom-poms and (2) stop by the cafeteria to sign up for the tryouts. Only then would she go to business class. Kamila's busy schedule requires her to keep track of many goals at once and to juggle them in her mind as the day passes, noting which tasks have been accomplished, which are left to be done, and, of those left to be done, which should be done next. Keeping track of goals, planning how to achieve them, and determining priorities all draw heavily on the central executive of working memory.

The French mathematician Edouard Lucas invented a game back in 1883 that requires many of these same planning and goal-setting abilities. The game is based on an ancient legend about a temple in India where a certain puzzle was used to develop mental discipline in young priests. A stack of 64 gold disks, each slightly smaller than the one beneath, were all stacked on a large pole. The young priest's assignment was to transfer all 64 disks from the first pole to a second pole and, finally, to a third pole by moving one disk at a time and only placing smaller disks on top of larger disks. According to the legend, if any priest ever solved the problem, the temple would crumble into dust, and the world would vanish. Perhaps one reason the world still exists today, thousands of years later, is that even if a very smart and quick priest were to move one disk per second, solving this task with 64 disks would take him 580 billion years.

Lucas called his simplified version, which was marketed as a board game, the Tower of Hanoi. At the game's start, the disks are placed on the leftmost of three pegs, arranged by increasing size from top to bottom, as in [Figure 9.8](#), which shows a small blue disk on top of a medium yellow disk on top of a large red disk. In order to move the disks properly and solve the puzzle, it helps to establish subgoals, such as getting the large red disk over to the rightmost peg, a maneuver that takes four moves.



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FIGURE 9.8 The Tower of Hanoi puzzle The objective of this task is to move all the disks from the leftmost peg to the rightmost peg, one disk at a time, without placing a larger disk on a smaller disk. The numbered sequence shows one way of doing this.

Description

Three pegs are attached to a common support, and there are three different colored discs. The steps are depicted as follows.

Step 1: The first peg has a red disc at the bottom, a yellow disc in the middle, and a blue disc at the top. The other two pegs are empty. A curved arrow points from the first peg toward the third peg.

Step 2: The first peg has the red and the yellow disc. The third peg has a blue disc. A curved arrow points from the first peg toward the second peg.

Step 3: The first peg has the red disc, the second has the yellow disc, and the third has the blue disc. A curved arrow points from the third peg toward the second peg.

Step 4: The first peg has the red disc. The second peg has the yellow disc at the bottom and the blue disc on top of it. The third peg is empty. A curved arrow points from the first peg toward the third peg.

Step 5: The first peg is empty. The second peg has the yellow disc at the bottom and the blue disc on top of it. The third peg has a red disc. A curved arrow points from the second peg toward the first peg.

Step 6: The first peg has a blue disc. The second peg has the yellow disc, and the third peg has the red disc. A curved arrow points from the second peg toward the third peg.

Step 7: The first peg has a blue disc. The second peg is empty. The third peg has the red disc at the bottom and the yellow disc on top of it. A curved arrow points from the first peg toward the third peg.

Step 8: The first two pegs are empty. The third peg has the red disc at the bottom, the yellow disc in the middle, and the blue disc on top.

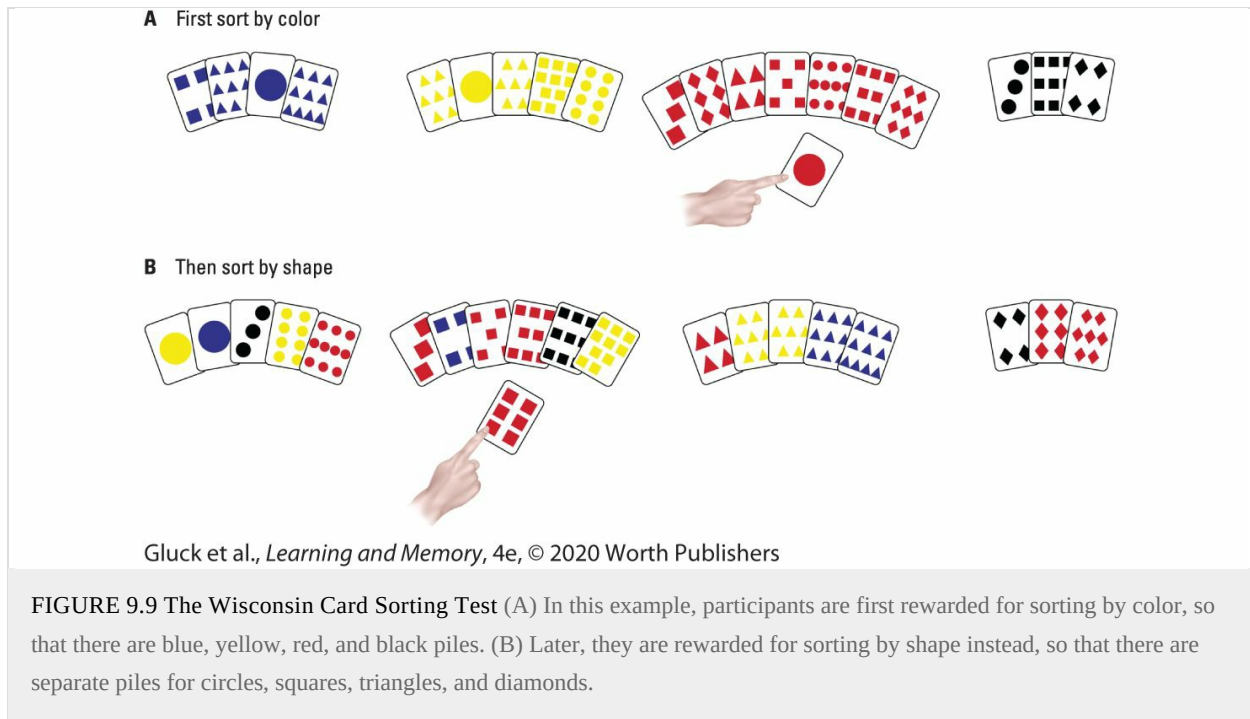
Solving the Tower of Hanoi puzzle requires a great deal of manipulation of working memory because you must remember at least three things at all times: (1) what subgoals have been accomplished, (2) what subgoals remain, and (3) what subgoal is the next to be addressed. After each move, some of these components will be updated and changed, while others will stay the same. This kind of goal-directed controlled updating of short-term memory is exactly the kind of task that places a heavy load on your central executive. It is hard enough to do with real disks and pegs, much less with sensory images and ideas that are only in your mind.

Task Switching

In business class, Kamila listens to the guest speaker with one ear. When the material in the lecture is familiar, she switches her attention to her biology reading. This kind of task switching requires the manipulation of working memory because Kamila must pay attention to the task she is doing at a given moment while at the same time monitoring external cues for information that may signal the need to switch to another task.

A commonly used procedure for studying task switching in the laboratory is the Wisconsin Card Sorting Test, in which people are shown cards with graphics that differ in three characteristics, or dimensions: number, color, and shape. One sample card might have three red circles, while another card might have one yellow triangle. On each trial, the participants are told to guess which of four piles a particular card should go on, and then they are told whether their choice is correct or not. In this way, the participants learn which of the three dimensions they should be paying attention to in order to sort the cards correctly. For example, a person might learn that it is correct for all the blue cards to go on one pile, all the yellow cards on another, and so forth, as illustrated in [Figure 9.9A](#). Later, however, after the person has discovered this sorting rule, the

task changes without warning, and the person receives positive feedback only when she sorts the cards by another rule. For instance, if the original rule was to sort by color, now the rule might switch to sorting by shape, making a pile for circles, a pile for squares, and so on, as shown in [Figure 9.9B](#). This task taps into people’s working memory and executive control because it requires not only learning a rule and keeping it in mind while they sort but also learning to change the rule and keep track of the new rule without confusing it with the old.



Description

Section A shows four sets of cards in blue, yellow, black, and red colors. The same colored cards are grouped together. Each set has cards with different shapes. A finger points to a card from the third set, with a red circle. In section B, the cards are grouped together based on their shapes. A finger points to a card from the second set, with red squares.

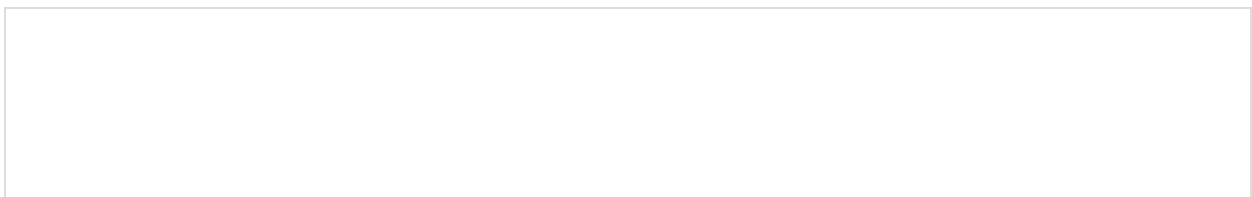
Stimulus Selection and Response Inhibition

While visiting Los Angeles, California, from his home in England, Trevor walks to an intersection and prepares to cross the street. He automatically begins turning his head toward the right to check for oncoming cars but quickly reminds himself that he is in the United States, and oncoming cars will come from the left, not the right. In this situation, information about his current context has inhibited Trevor’s ingrained, reflexive response and redirected his attention.

Trevor's central executive functioning allows him to inhibit a habitual response that he has developed and shift his attention to an alternative, context-specific rule ("look left when crossing streets in the United States") that he must remember—perhaps through repeated rehearsal when walking through Los Angeles—as long as he remains in the United States. Given how difficult it is to suppress such well-learned reflexive responses, it is not surprising that many London intersections have "Look Right" painted on the curb in an attempt to reduce the number of American and European visitors being injured after looking the wrong way and walking into oncoming traffic.

[Don Norman and Tim Shallice \(1986\)](#) proposed that situations like these highlight the distinction between two types of processes—automatic ones and willed, or controlled, ones—that occur in parallel, competing to influence our behaviors. Automatic processes are triggered by situational cues, such as approaching a street, which to a British person like Trevor elicits an overlearned quick turn to the right to look for oncoming cars. These automatic responses are deeply ingrained and reflexive and typically occur with minimal conscious awareness; they do not generally interfere with other concurrent activities (like talking to a friend with whom we are walking). In contrast, willed, or controlled, actions are mediated by what Norman and Shallice described as the *supervisory attentional system*, which modifies behavior when the automatic responses are inappropriate, inhibits automatic routines in favor of more appropriate behaviors, and changes the prioritization of cues for attention; all of these can be viewed, in the terminology of Baddeley's model, as functions of the central executive.

A well-known test of how well a person's control processes can direct attention to stimuli and inhibit inappropriate automatic responses is the Stroop task ([Stroop, 1935](#)), which creates a laboratory situation analogous to Trevor's process of adapting to the traffic patterns in the United States. The Stroop task consists of a series of names of colors, each printed in a color that is *different* from the color being named ([Figure 9.10](#)). The word "green" might be printed in red ink, the word "blue" in green ink, the word "black" in blue ink, and so forth. The task is to look at each word in turn and say the color it is printed in, ignoring what the word happens to say.



Green
Blue
Black
Red
Orange
Purple
White
Yellow

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FIGURE 9.10 The Stroop task The names of colors are printed from top to bottom, each in a color that does not correspond to the name. The task is to recite the colors that the words are printed in (color of ink) without being distracted by what the words say.

Description

The names of colors and the color in which they are printed are as follows. Green-printed in red, Blue-printed in green, Black-printed in blue, Red-printed in black, Orange-printed in green, Purple-printed in red, White-printed in black, and Yellow-printed in blue.

Because people usually respond automatically to a written word by reading it, the Stroop task is very difficult to perform smoothly. Try it, and you will see how hard it is to overcome the almost irresistible urge to read what the words say. To perform the task rapidly, you must inhibit your automatic impulse to read the words and instead keep a context-specific goal in your working memory to remind you of the task at hand: to attend to the ink color alone, much as Trevor must keep in mind the rule to “look left for cars” when he is crossing the road in the United States. Thus, the Stroop task requires inhibiting currently inappropriate automatic reflexive responses while attending to the task-specific aspects of a stimulus.

The Stroop task illustrates the fundamental competition within our brains over the control of our behaviors and thoughts. The various inputs competing for our attention at any given moment include both bottom-up processes that are stimulus driven (potentially eliciting automatic reflexes and well-learned responses) and top-down processes that are goal directed and require selective attention to specific aspects of our environment. Although the simple hardwired, reflexive responses are useful for many situations that require rapid responding and minimal conscious effort, they are insufficient for dealing with the more complex facets of our dynamic world, where long-range planning, goals, and the ability to foresee future consequences are necessary ([Miller & Wallis, 2003](#)).

Test Your Knowledge

Varieties of Working Memory and Cognitive Control

For each of the following scenarios (1 through 4), indicate which facet of working memory (A through D, below) is in evidence. ([Answers appear at the end of the chapter.](#))

1. Texting while driving
2. Remembering not to wake up in the middle of night and call out the name of your old girlfriend while you are sleeping with your new girlfriend

3. Remembering to pick up donuts, toilet paper, and seltzer water at the convenience store while you make your way up and down the aisles
4. Preparing for your friends a meal consisting of an entrée, a salad, and two side dishes and having all the dishes ready to serve at the same time

CHOICES:

- A. *Controlled updating of short-term memory buffers*
- B. *Setting goals and planning*
- C. *Task switching*
- D. *Stimulus selection and response inhibition*

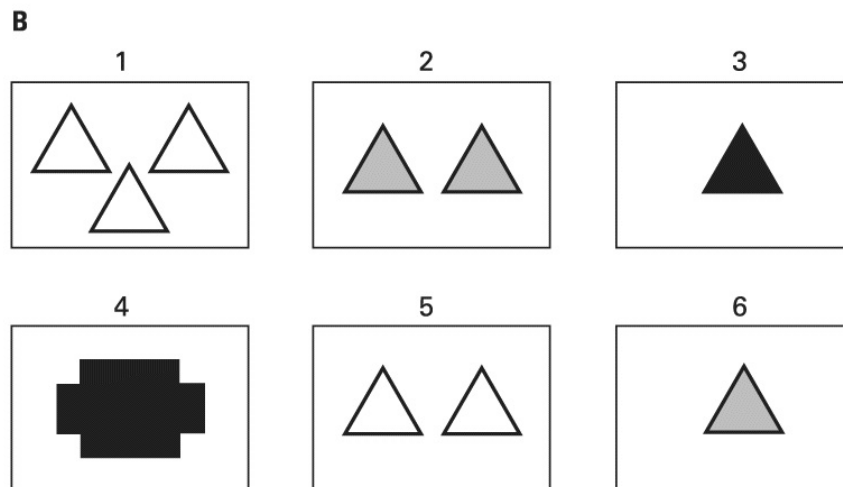
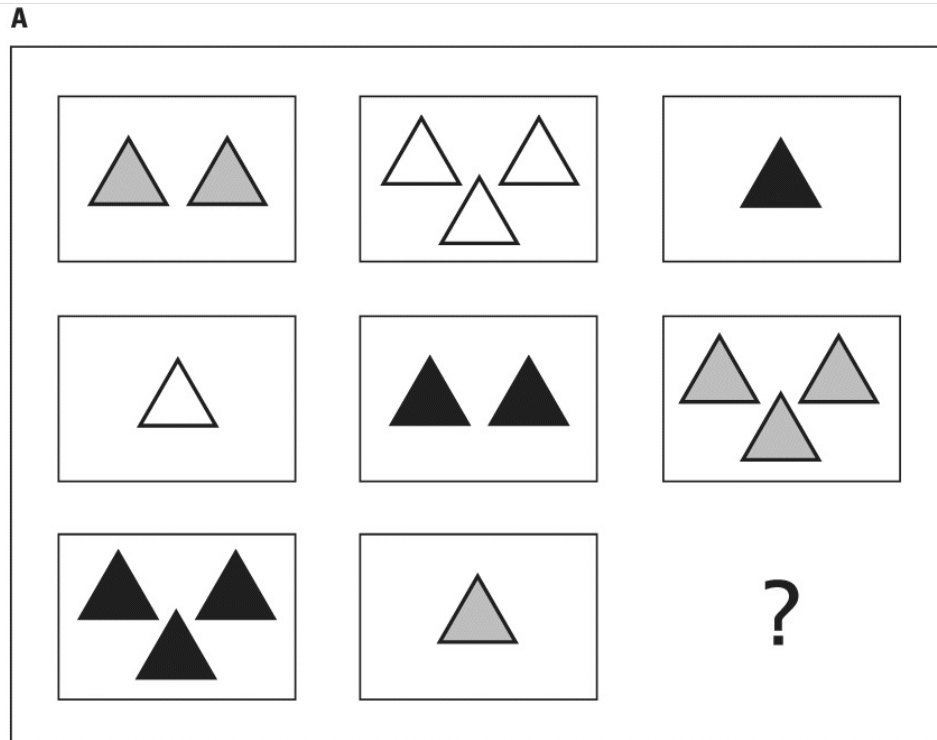
Are Working Memory and Cognitive Control the Keys to Intelligence?

Intelligence, defined as the capacity for learning, reasoning, and understanding, is a familiar enough term, but the concept is often poorly understood. Intelligent people are frequently described as “quick,” but a growing body of research suggests that intelligence has less to do with the brain’s processing speed and more to do with executive control of working memory.

Assessing students’ working memory using a delayed recall task, Meredyth Daneman and Patricia Carpenter found a strong correlation between working-memory scores and verbal SAT tests of reading comprehension ([Daneman & Carpenter, 1980](#)). The latter are often viewed as an approximate indication of intelligence, although others have argued they are simply an indicator of expected performance in college.

Evidence of a relationship between working memory and intelligence does not depend only on verbal intelligence, however. Carpenter and her colleagues used puzzles based on standard nonverbal tests of intelligence called the Raven Progressive Matrices. These puzzles employ two-dimensional visual analogy problems in which the participant is directed to select the design that completes each pattern. The illustrative example shown in [Figure 9.11](#) is a three-by-three array of geometric figures with a blank space in the lower-right corner. Participants must pick which of the six alternatives at the bottom best fits the pattern. What is the pattern? Note that the figures vary on two dimensions: the color (black, gray, or white) and the number of triangles (1, 2, or 3). Moreover, no row or column has two figures with the same number of triangles or the same color. To complete this pattern, the figure in the lower right would have to be white (there

is no white figure in the third row or third column) and contain two triangles (there is no figure with two triangles in the third row or third column). Thus, the correct answer is #5, two white triangles.



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FIGURE 9.11 Raven Progressive Matrix Test of Nonverbal Intelligence In this example, subjects are shown a three-by-three array of eight geometric figures and a blank space, in the lower-right corner, where the ninth figure belongs. They must pick which of the six alternatives shown at the bottom best fits the pattern. (The correct answer is #5.)

Description

Section A shows 8 smaller rectangles arranged in 3 rows within a larger rectangle, each containing different shapes and shades as follows. The eighth smaller rectangle is replaced by a question mark symbol.

Row 1: rectangle 1- two gray triangles, rectangle 2- three white triangles, two arranged on top and one between them at the bottom, rectangle 3- a black triangle.

Row 2: rectangle 1- a white triangle, rectangle 2- two black triangles, rectangle 3- three gray triangles, two arranged on top and one between them at the bottom.

Row 3: rectangle 1- three black triangles, rectangle 2- a gray triangle, rectangle 3- question mark symbol.

Section b shows 6 rectangles numbered from 1 through 6, arranged in 2 rows within a larger rectangle, each containing different shapes and shades as follows. Rectangle 1- three white triangles, two arranged on top and one between them at the bottom, rectangle 2- two gray triangles, rectangle 3- a black triangle, rectangle 4- a 9 sided shape shaded in black, rectangle 5- two white triangles, rectangle 6- a gray triangle.

An enormous range of difficulty can be introduced into this kind of task by increasing the complexity of the patterns or the number of components. Carpenter and colleagues showed that being able to juggle many rules in one's head is correlated with scoring high on nonverbal tests of intelligence. These types of results suggest that general intelligence, as detected by intelligence tests, is not just a matter of thinking or responding quickly. Rather, general intelligence appears to be associated with a strong working memory, especially in terms of the control and manipulation of larger numbers of rules, concepts, goals, and ideas.

Given how critical an optimally functioning working memory is to our daily lives, many people wish they could make better use of theirs. For tips on how this might be possible, see the [“Learning and Memory in Everyday Life” box](#).

LEARNING AND MEMORY IN EVERYDAY LIFE

Give Your Working Memory a Break

Because working memory and executive control are fundamental to our higher cognitive abilities, it is natural to wonder what can be done to improve them. A key finding from research on working memory is that our visuospatial and verbal working memories are independent, and each has a limited capacity. You can probably keep only about 3 to 5 items in either store at any one time, which makes remembering a 10-digit phone number a bit tricky. One possible solution when you need to remember a long list of items is to make the independence of visuospatial and verbal memory stores work *for* you rather than against you. For example, if you need to remember several words at once, such as people's names, consider converting some of the words into pictures so that both memory buffers can share the daunting task.

The biggest drain on our working memory comes from multi-tasking, or attempting to accomplish several goals at once.

How often do you talk on the phone, listen to music, and surf the Internet all at the same time? You can bet that your dorsolateral prefrontal cortex is working overtime when you do. Of more concern is multi-tasking during dangerous tasks, such as driving in traffic. Have you ever seen someone try to read a newspaper, apply nail polish, or talk on a cell phone while behind the wheel? Unfortunately, traffic accidents often result from people's attempts to multi-task while driving. For this reason, many states have banned cell-phone use while driving, especially the use of handheld phones.

An overloaded working memory impairs the ability to accurately monitor and evaluate our cognitive functioning. You might think your driving is just fine or that you can absorb the main ideas in your professor's lecture while you work on other projects (like Kamila, who studies biology during business class), but research has shown that you are probably operating at a lower level than you think you are. Focusing on one task at a time greatly improves the ability to use working memory effectively. In contrast, high levels of stress reduce the working-memory span and the ability to concentrate and focus cognitive control. Some research has suggested that stress elevates dopamine levels in the prefrontal cortex, impairing its ability to efficiently monitor and update information.

Why tax your working memory if you don't need to? Maybe it's time to shut off that cell phone, grab a pad of paper (think of it as a third working-memory buffer), and start writing things down.

Interim Summary

- Transient memories are temporary representations of information. They are found in the first two stages—sensory memory and short-term, or working, memory—of the Atkinson–Shiffrin model of the stages of memory storage. Short-term memories can be maintained by active rehearsal but are easily displaced by new information or distractions.
- Alan Baddeley characterized working memory as consisting of two independent short-term memory buffers—the visuospatial sketchpad, which holds visual and spatial images, and the phonological loop, an auditory memory store that uses internal speech rehearsal—along with a central executive.
- In Baddeley's model, the central executive is responsible for manipulating memories in the two buffers by, for example, adding and deleting items, selecting items to guide behavior, and retrieving information from and storing it in long-term memory.
- The manipulation and use of working memory for cognitive control can be measured through neuropsychological tests that assess such functions as controlled updating of short-term memory, setting goals and planning, task switching, and stimulus attention and response inhibition.
- Earlier conceptions that viewed working memory (and short-term memory) as being in a different place than long-term memory have now evolved to a more nuanced “single unitary-storage” view of memory, which sees these different facets of memory as being different states of memory rather than in different places.

9.2 Brain Substrates

Could the frontal lobes contain the brain's central executive, in charge of working memory, reasoning, and other cognitive functions? There is a growing consensus that the prefrontal cortex, with its extensive connections to more posterior regions of the brain, is critical for initiating executive functions and cognitive control and that it does so by integrating external and internal information needed to guide complex behaviors ([Miller & Wallis, 2003](#)). If this is the case, how do the frontal lobes mediate working memory and cognitive control? How does working memory function in the brain? These questions drive current research as scientists seek to understand to what extent psychological theories, such as Baddeley's model, correspond to how working memory is organized in the brain. Does the brain in fact possess a distinct, recognizable working memory system, or is working memory simply the transient and current activity of the brain's memory system in general?

Frontal-Lobe Anatomy and Consequences of Frontal-Lobe Damage

Studies of animals and humans implicate the frontal lobes—especially the [prefrontal cortex \(PFC\)](#), the most anterior (farthest forward) section of the frontal lobes—as being critical for working memory and executive control. Since the beginning of modern brain research in the mid-1800s, the intellectual functions of planning, foresight, decision making, and related behaviors have been associated with this brain region ([Markowitsch, 1992](#)).

prefrontal cortex (PFC)

The frontmost (anterior) part of the frontal-lobe cortex, essential for working memory and executive control.

In humans, the prefrontal cortex encompasses approximately one-third of the cerebral cortex ([Goldman-Rakic, 1987](#)). Cats and many other mammals, on the other hand, get by with frontal lobes that occupy less than 4% of their cerebral cortex. [Figure 9.12](#) compares the relative sizes of the prefrontal cortices in several mammalian species, showing the human prefrontal cortex as compared with the prefrontal cortices in cats, rhesus monkeys, and chimpanzees. Because the prefrontal cortex occupies a markedly larger proportion of the cerebral cortex in humans than in many other mammals, many people have assumed that this is precisely what makes us human. More recent studies, however, have shown that the frontal cortices of humans are not disproportionately larger than those seen in great apes such as gorillas and orangutans. This

discovery suggests that the human cognitive abilities attributed to large frontal lobes may also reflect characteristics other than size, such as more complex interconnections or specialized subregions found within the human frontal cortex ([Semendeferi, Lu, Schenker, & Damasio, 2002](#)).

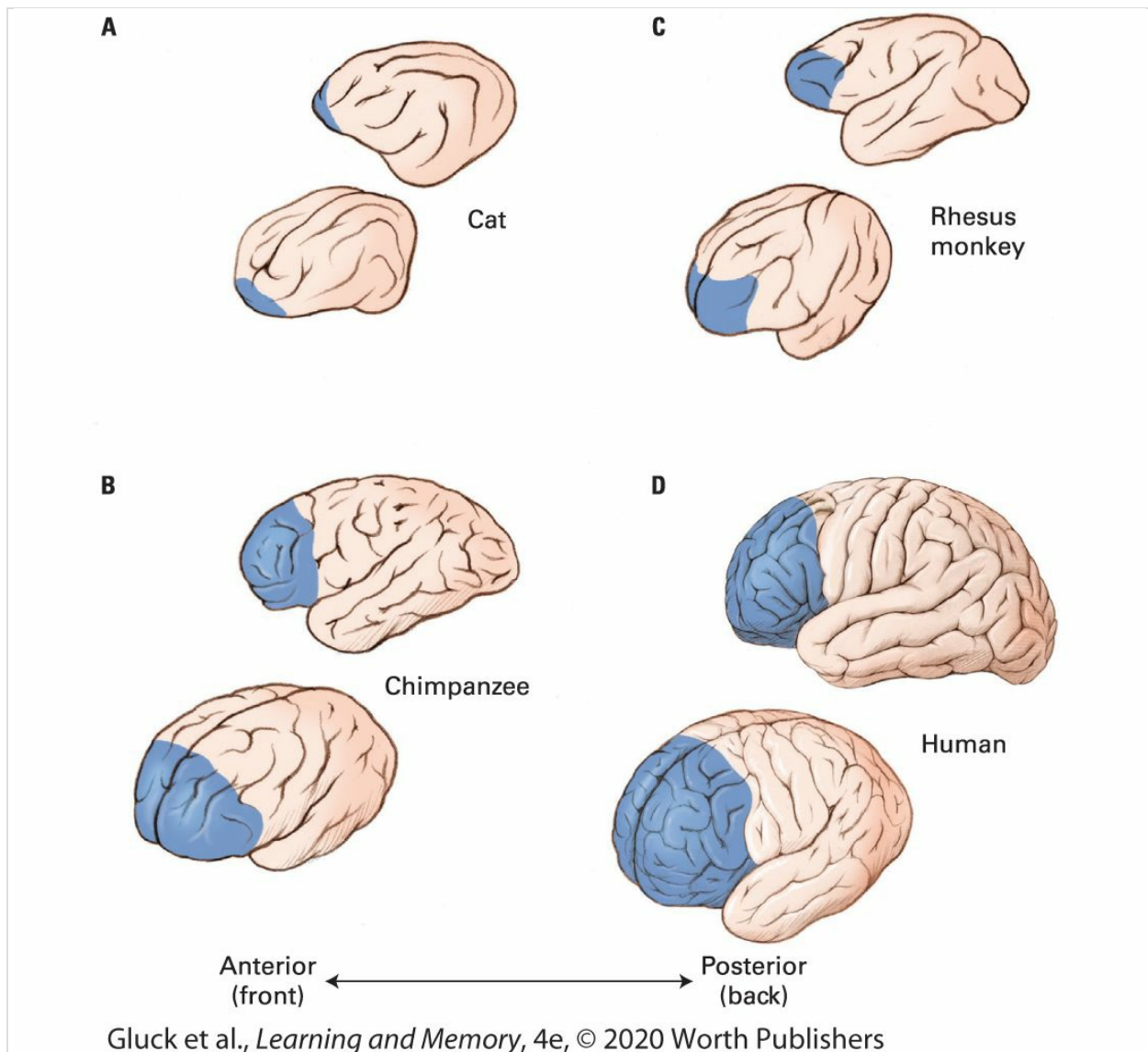


FIGURE 9.12 Comparative frontal-lobe anatomy These drawings show the relative sizes of the prefrontal cortices in different mammals.

Information from [Fuster, 1995](#).

Description

These organisms can be arranged in increasing order of the size of their prefrontal cortices as follows. Cat, Rhesus

monkey, Chimpanzee, and Human. A double-headed arrow at the bottom shows the anterior, or front, and posterior part, or the back.

Behavior Changes Following Frontal-Lobe Damage

Some insights into the role of the prefrontal cortex have come from observing the behaviors of people with frontal-lobe damage. During World War I, for example, the many soldiers returning from battle with head wounds provided evidence of a link between the frontal lobe and intellect. A study of 300 such veterans showed that those who had frontal-lobe damage were the most impaired in higher intellectual abilities ([Pfeifer, 1922](#)).

One noted characteristic of frontal-lobe damage is a loss of ability to plan and to organize. Wilder Penfield, a famous neurosurgeon from the mid-twentieth century who did pioneering work on brain mapping, described the changes in his own sister after a large tumor was removed from her frontal regions. She had been an accomplished cook, but after the surgery she lost all ability to organize her cooking; she would move haphazardly from dish to dish, leaving some dishes uncooked while others burned ([Miller & Wallis, 2003](#)).

Consider Elliot, a successful and happily married accountant, who had always been viewed by others as reliable and responsible. In his late thirties, he developed a large tumor in his frontal lobes. Surgeons were able to remove the tumor and save his life. However, the operation severely damaged his frontal lobes ([Damasio, 1994](#); [Eslinger & Damasio, 1985](#); [Miller & Wallis, 2003](#)). Soon after the surgery, he divorced his wife, remarried and divorced again, lost touch with most of his friends and family, got involved in corrupt business deals, and was soon bankrupt. The formerly responsible and cautious Elliot had become impulsive and easily swayed and distracted by momentary whims, and he retained little of his previous ability to organize and plan.

Penfield's sister and Elliot were both behaving as we might expect in the absence of a cognitive-control system. They were no longer guided by long-term goals or task-specific constraints. Rather, they appeared to be ruled by their reflexive and automatic impulses and to have little capacity for foresight and planning. For this reason, people like Penfield's sister and Elliot are described as having [dysexecutive syndrome](#), a disrupted ability to think and plan ([Duncan, Emslie, Williams, Johnson, & Freer, 1996](#)). As you will read next, patients with frontal-lobe

damage routinely exhibit deficits in both executive function and working memory.

dysexecutive syndrome

A disrupted ability to think and plan.

In addition to being damaged by tumors and surgery, frontal lobes can be damaged by strokes or blunt trauma to the front of the head—or, as often happens, from a rapid deceleration (as in a car crash) in which the frontal lobes compress against the front of the skull. Professional (and even some high school) football players, who routinely head-butt other players and experience rapid deceleration, also show a pattern of frontal-lobe damage and associated cognitive problems ([Amen et al., 2011](#)).

Deficits in Working Memory Following Frontal-Lobe Damage

People with damage to the frontal lobes show deficits on all of the working-memory and cognitive-control tasks described in [Section 9.1](#). For example, they have great difficulty updating working memory in the *N*-back task, as well as performing self-ordered tasks that require frequent updating to recollect items that have been previously chosen ([Petrides, 2000](#)). Patients with frontal-lobe damage are also often impaired at tasks that tap short-term memory span, including digit-span tasks, in which they may fail to recall even a short series of numbers ([Janowsky, Shimamura, Kritchevsky, & Squire, 1989](#)). Other studies of these patients have shown similar impairments in short-term memory for colors, shapes, and object locations ([Baldo & Shimamura, 2000](#); [Ptito, Crane, Leonard, Amsel, & Caramanos, 1995](#)).

In addition, as you might expect, patients with frontal-lobe damage show deficits in neuropsychological tests such as the Tower of Hanoi, which assesses planning abilities and requires a person to maintain and link multiple subgoals to achieve the final desired goal. On the Tower of Hanoi task, patients like Elliot or Penfield's sister move the disks around aimlessly, without a clear plan for getting the disks from the first peg to the last.

The ability to shift appropriately from one task to another is a central feature of executive control. Thus, task-switching test procedures, such as the Wisconsin Card Sorting Test described in [Section 9.1](#), provide another means of assessing frontal-lobe function. One study asked participants to monitor two streams of simultaneously presented stimuli, a series of letters on the

left and a stream of digits on the right ([Duncan et al., 1996](#)). At the beginning of the experiment, the participant was to read aloud the letters on the left. Later, when a signal cue was sounded, the person was supposed to switch to the other stream and begin reporting the digits. Later still, the signal would sound again as a sign that the participant should switch back to reading the letters. Although patients with frontal lesions had no trouble complying with the first part of the experiment, they had great difficulty switching between the left and right streams on cue.

When the Wisconsin Card Sorting Test (see [Figure 9.9](#)) is used in assessment of frontal-lobe function, patients with frontal-lobe damage have no problem learning an initial sorting rule, such as to sort by color. Later, however, when they must learn a new rule for sorting—say, by shape—these patients are severely impaired at making the transition. They show **perseveration**, which means they fail to learn a new rule and, instead, persist in using an old rule despite repeated feedback indicating that the old rule is no longer correct. The severe deficits in task shifting associated with frontal-lobe damage suggest that purposeful shifts in processing may be especially demanding of executive-control operations mediated by the frontal lobes ([Delis, Squire, Bihle, & Massman, 1992](#); [Owen, Evans, & Petrides, 1996](#)).

perseveration

A failure to learn a new response, especially as demonstrated by continued adherence to an old response rule that is no longer valid.

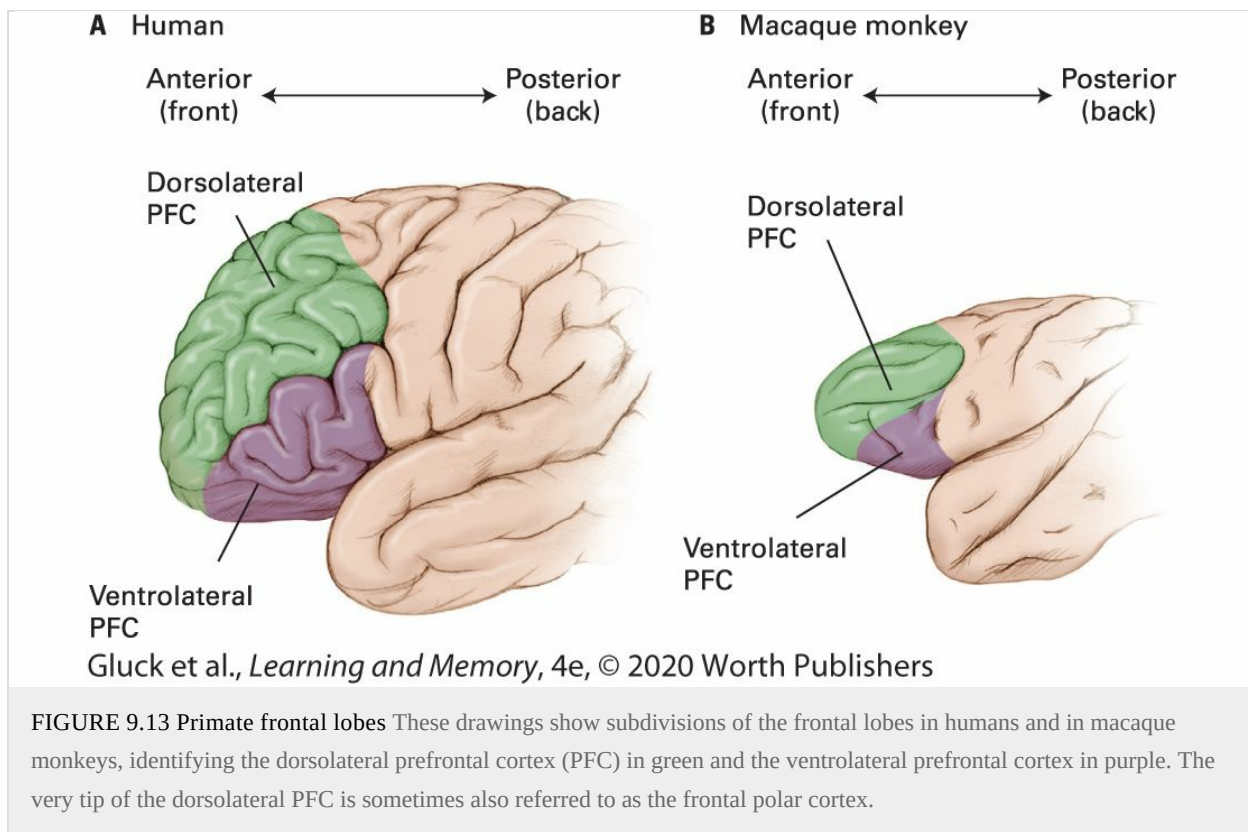
Carlyle Jacobsen (mentioned in [Section 9.1](#)) was an early and important researcher in this area, who conducted animal studies in the early 1930s that implicated the frontal cortex in working memory ([Jacobsen, 1936](#)). Specifically, he looked at the effects of lesions in different parts of the brain on delayed spatial-response learning in monkeys. In these studies, monkeys were permitted to observe food being placed either in a location on the left or on the right of a surface outside their cages. After a delay, during which the monkeys were not able to see the food, the monkeys were required to point to where the food had been placed. Jacobsen demonstrated that only monkeys with prefrontal lesions were impaired at responding correctly. Based on these results, he argued that an animal's frontal lobes are critical for maintaining an internal representation of information in working memory over a delay prior to making some response.

One limitation of this early work was the relative crudity of Jacobsen's surgical techniques by modern standards: he removed a rather large part of the prefrontal cortex that likely included many specialized subregions. More recent research has shown that different subregions of the

prefrontal cortex participate in different aspects of working-memory function.

Divisions of the Prefrontal Cortex

The primate prefrontal cortex can be divided into three main regions: the *orbital prefrontal cortex*, the *medial prefrontal cortex*, and the *lateral prefrontal cortex*. The lateral prefrontal cortex can be further subdivided into two components: the **dorsolateral prefrontal cortex (DLPFC)** on the top and the **ventrolateral prefrontal cortex (VLPFC)** below it, as shown in **Figure 9.13**. (The orbitofrontal cortex is not visible in these drawings because it lies ventral to—that is, below—the regions shown; the medial prefrontal cortex is not visible because it is inside the regions shown, tucked away above and behind the orbital region; see, however, [Figure 5.9](#).) Both the orbitofrontal and medial prefrontal cortices are implicated in many memory and cognitive-control functions, especially goal-driven behavior and planning; but they are less involved in working memory than are the lateral regions of the prefrontal cortex and thus will not be discussed further in this chapter. Much of what we know about the role of the DLPFC and VLPFC in working memory comes from recordings of brain activity in humans and monkeys. Data from such studies will be discussed next.



Description

The size of the dorsolateral P F C and Ventrolateral P F C in the human brain is larger than that of a macaque monkey. The dorsolateral P F C is labeled in the anterior part of the brain, on top of the ventrolateral P F C. The dorsolateral P F C is highlighted in green and ventrolateral P F C is highlighted in purple. Two double-headed arrows on top of the two diagrams show, anterior part as front and posterior part as back.

dorsolateral prefrontal cortex (DLPFC)

The left and right sides of the topmost part of the prefrontal cortex (PFC).

ventrolateral prefrontal cortex (VLPFC)

The lower left and right sides of the PFC.

Studies of Frontal Brain Activity During Working-Memory Tasks

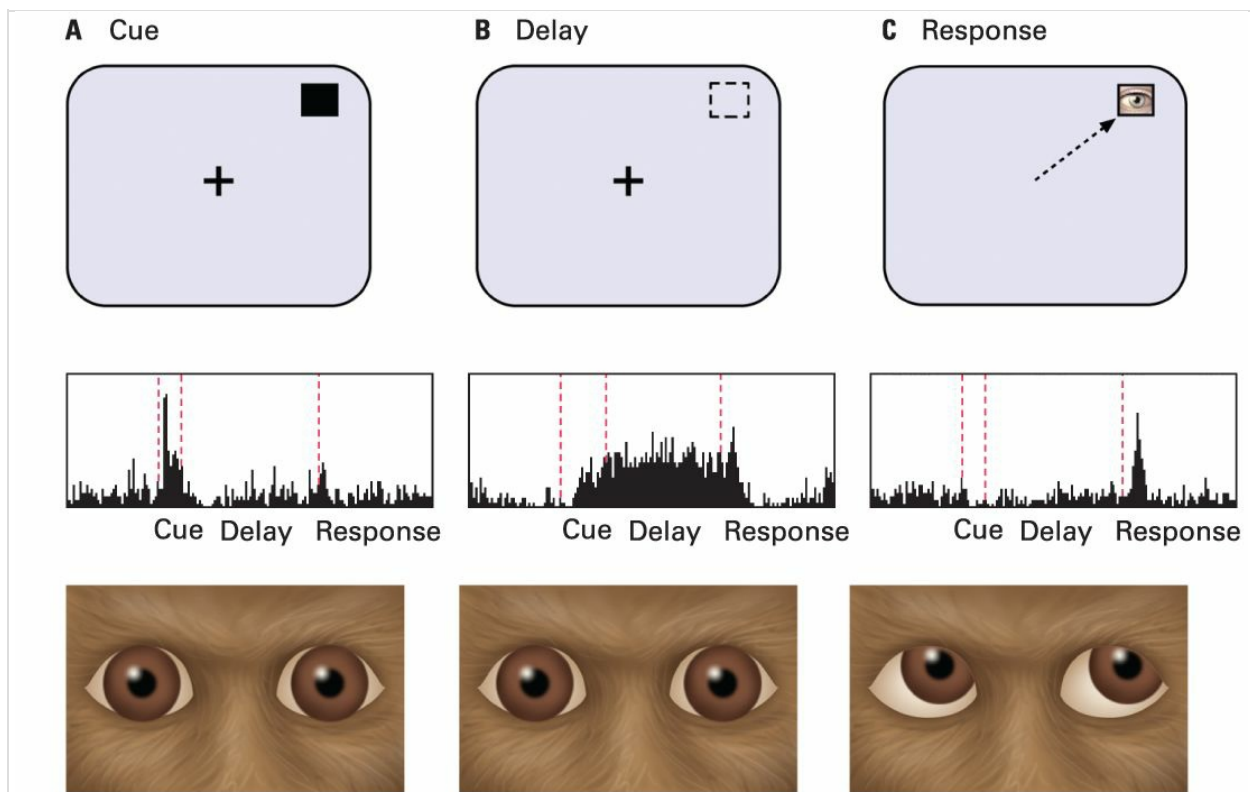
Guided by lesion studies suggesting that the prefrontal cortex plays a role in working memory, researchers in the early 1970s began to record prefrontal-cortex neural activity during working-memory tasks ([Fuster & Alexander, 1971](#); [Kubota & Nikin, 1971](#)). In a study by Joaquin Fuster, using a delayed-response task similar to the one used in the Jacobsen studies described earlier, rhesus macaque monkeys were required to remember either where they had seen a target object or what object they had previously seen ([Fuster & Alexander, 1971](#)). The researchers found that some prefrontal-cortex neurons fired only during a delay period and only when the animals were required to maintain information about the spatial location of a particular object. This suggested to them that the prefrontal cortex might be “holding in mind” information needed to make a later response. If so, then the neural activity in the prefrontal cortex was acting as a temporal bridge between stimulus cues and a contingent response concerning object location in order to facilitate the linking of stimulus and response events across time.

Such activity appears to be a key component of sensorimotor behaviors that span delays ([Fuster, 2001, 2003](#)). Persistent activity of this type in the DLPFC during blank memory intervals (delays, when no external stimulation is present) is a strong argument implicating the DLPFC as a critical brain region supporting the maintenance of items in working memory ([Curtis & D’Esposito, 2006](#)).

Instead of requiring an animal to pick up an object or point to a location, some experiments simply track the animal’s gaze. Eye-tracking technology offers well-controlled methods for

testing spatial and object working memory in animals. Patricia Goldman-Rakic of Yale University Medical School, one of the pioneers in working-memory research, used this technology in a series of highly influential studies of primate working memory.

In her studies, Goldman-Rakic trained monkeys to fixate on a central spot of a display like the one shown in [Figure 9.14A](#) (top row). The monkeys maintained their fixation on the central spot while a square cue was presented at one of eight locations around the edge of the display (e.g., the upper-right corner, as in [Figure 9.14A](#)). After the cue was removed, the monkeys waited during a delay period of several seconds ([Figure 9.14B](#)), as they were trained to do; then, at a certain signal, they responded by moving their gaze to the cue's former location ([Figure 9.14C](#)). Moving the gaze to the correct location resulted in a reward.



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FIGURE 9.14 The spatial delayed-response eye-gaze task (A) The monkey fixates on a central spot on the screen while a cue flashes in the upper-right corner. (B) During a delay period, the cue disappears, and the monkey remains fixated on the central point. (C) Finally, when the central spot turns off, the monkey looks where the cue previously appeared. (For clarity, the monkey is shown in mirror image in the figure so we can see it gaze in the direction of the stimulus shown above.)

Data from [Funahashi, Bruce, & Goldman-Rakic, 1989](#).

Description

Three screens are shown on top labeled as A-Cue, B-Delay, and C-Response, respectively. A graph and a schematic correspond to each screen. The labels on the horizontal axis of the graph read, cue, delay, and response. The information presented is as follows.

A-Cue: the screen shows a plus sign at the center and a rectangular cue at the top right corner. The curve in the graph peaks during cue, and falls to a minimum during delay and response periods.

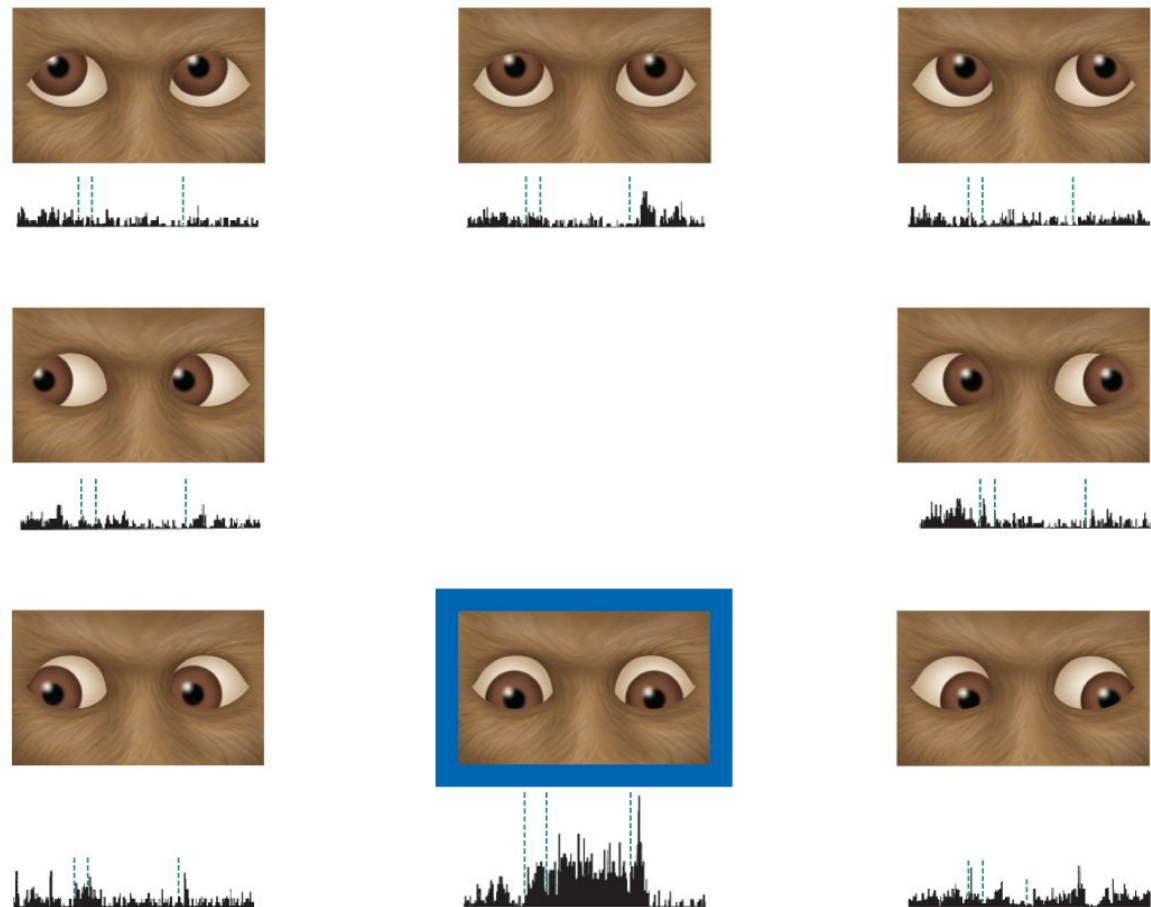
B-Delay: the screen shows a plus sign at the center, and a dashed rectangle is shown in place of the cue. The curve in the graph peaks during delay time, and falls to a minimum during cue and response periods.

The eyeball of the monkey remains focused toward the central plus sign during cue and delay periods.

C-Response: the screen shows an arrow pointing toward the top right corner of the screen, where the cue was flashed initially. The eyeball of the monkey focusses on the top right corner of the screen.

In electrophysiological recordings of these tasks, Goldman-Rakic and colleagues found that some of the neurons in the dorsolateral prefrontal cortex fired only while the animal was remembering the stimulus location ([Funahashi, Bruce, & Goldman-Rakic, 1989](#)). As shown in the electrical recordings in the middle row of [Figure 9.14](#), certain neurons in the prefrontal cortex fire during presentation of the cue itself ([Figure 9.14A](#); note the increase in firing during presentation of the cue in the area between the two red lines on the left). Others fire only during the delay period ([Figure 9.14B](#); note the increase in firing during the delay period), and still others fire during the response ([Figure 9.14C](#); note the increase in firing to the right of the rightmost red line). Each of the three recordings in [Figure 9.14](#) shows a different neuron.

Most interesting of all is that the “delay” neurons were individually tuned to different directional eye movements. For example, one neuron might code for a movement to the right, while another neuron might code for a downward movement, and so on. [Figure 9.15](#) shows the strong response of a particular neuron when the cue was located at the bottom center of the screen (bottom-center graph) compared with the inhibition of its electrical activity when the cue was in the opposite location (top-center) and compared to the moderate activity when the cue was at other locations.



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FIGURE 9.15 Response of one prefrontal cortex neuron during the delayed-response eye-gaze task Electrophysiological activity of the neuron during the cue, delay, and response periods of the task when the cue was presented at different locations. Note the strong response when the cue was at the bottom-center location (indicated by the dark blue outline) compared with the inhibited activity when the cue was presented at the top-center location. (For clarity, the monkey is shown in mirror image in the figure so that we see it gaze in the same direction as the stimulus being shown to it.)

Data from [Funahashi et al., 1989](#).

Description

Eight schematics show the eyeball of a monkey fixating on different spots on the screen, where the cue is flashed. A graph corresponding to each schematic depicts the response. The response of the monkey is maximum, in the graph corresponding to a schematic highlighted in blue, in which the eyeball of the monkey remains fixated at the bottom-center of the screen.

The strong firing seen in [Figure 9.15](#) during the delay for the trial in which the cue was at bottom center could represent one of two things: it could be a memory for where the cue had appeared, or it could be an anticipatory coding for the later movement of the eye gaze to that location. To

distinguish between these alternatives, the researchers conducted an experiment in which the monkeys were trained to move their eyes to the location *opposite* the cue. In that study, about 80% of the delay neurons seemed to encode where the target had been (regardless of the eye-gaze response), whereas the other 20% seemed to encode the intended movement. These results suggest that the neurons of the dorsolateral prefrontal cortex that fire during the delay are encoding a combination of sensory and movement-response information.

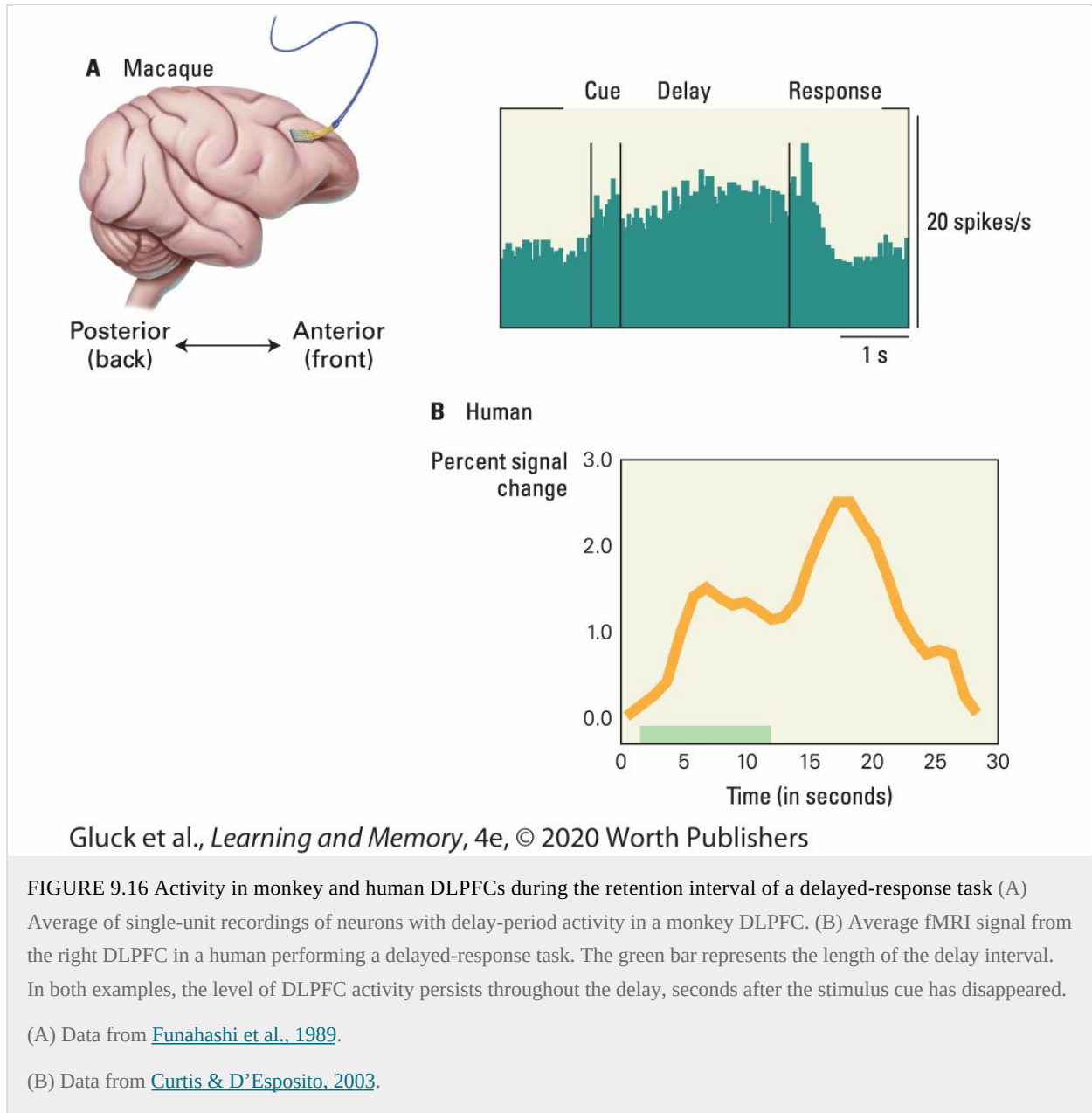
The monkeys did quite well in this delayed-response task, but they never performed it with 100% accuracy. Occasionally, they would make an error and move their eyes to the wrong position. Was it just a motor mistake, or was the PFC itself confused about the correct answer? The researchers found the latter to be true: the electrophysiological recordings predicted when a monkey was going to make an error because the “wrong” neurons fired in the DLPFC.

Sustained neuronal activity during the delay period is not limited to the DLPFC. Similar sustained activity can also be seen in the relevant primary and secondary sensory and motor regions in the temporal and parietal lobes, located in the more posterior regions of the brain. These regions are reciprocally connected to the PFC.

If the sensory and motor cortices can sustain activity during a delay (assuming that sustained activity is indeed critical for working memory), why should the PFC be necessary for working memory to function? Earl Miller argued that the key “cognitive” contribution of the PFC to working memory is its ability to sustain activity *despite distractions* ([Miller, 2000](#)). To test his hypothesis, Miller and colleagues trained monkeys to maintain the visual memory of an object throughout a delay period filled with visually distracting events ([Miller, Erickson, & Desimone, 1996](#)). They found that activity in the posterior visual cortical areas was easily disrupted by the distractors.

In contrast, the corresponding DLPFC activity remained robust despite distractions. The ability of the PFC to provide focused control over working memory is consistent with lesion data demonstrating that one salient consequence of prefrontal-cortex damage—in humans as well as in monkeys—is a high degree of distractibility. In addition, event-related fMRI studies in humans have shown persistent activity in the DLPFC during the retention interval of delayed-response tasks. This is illustrated in [Figure 9.16](#), which shows similar patterns of activity from

both monkey and human DLPFCs during the delay period in a task in which subjects were required to withhold responding to a cue, as in the studies of Goldman-Rakic and others described previously.



Description

Section a: the schematic shows the exterior view of a Macaque brain with a wired electrode attached to its anterior part. A double-headed arrow points toward the posterior and the anterior parts of the brain.

Section b: a graph depicts cue, delay, and the response of the Macaque brain. 20 spikes per second are recorded.

Section c: the graph plots percentage signal change in a human brain on the vertical axis against time in seconds on the horizontal axis. The readings on the horizontal axis range from 0 to 30 in increments of 5 and the readings on the vertical axis range from 0 to 3 in increments of 1. The curve originates from (0,0), attains a smaller peak at (7,1.75), descends to a point at (14,1.2), after which it attains a second peak at (17,2.6). The curve descends gradually after that, ending at (29,0). The time period corresponding to 0.2 and 12 seconds and 0 percent signal change is highlighted in green.

A related view concerning the importance of persistent DLPFC activity in controlling behavior is that top-down signals from the DLPFC have a diverse range of roles, depending on which brain region is the recipient of the signal ([Curtis & D'Esposito, 2003](#)). That is, the DLPFC may always be performing the same general function: control. However, when DLPFC control is exerted over different brain regions, the effects on behavior may be quite different. Thus, top-down signals from the DLPFC might enhance and maintain internal representations of relevant sensory stimuli in different posterior sensory and motor centers until those representations are required for subsequent actions and decisions.

Mapping Executive Processing and Working Memory onto PFC Anatomy

The lesion, electrophysiological, and imaging studies reviewed here leave two critical questions unanswered: (1) how are the frontal lobes organized, and (2) how does working memory actually work? In particular, are there different physical systems (or anatomical regions) in the brain for executive processes (memory manipulation) and rehearsal processes (memory maintenance), as suggested by Baddeley's model? That is, does the *functional* distinction between manipulation and rehearsal proposed by Baddeley correspond to an actual *anatomical* distinction between distinguishable brain regions? Also, are there anatomical distinctions associated with the two material-specific rehearsal stores—the visuospatial sketchpad and the phonological loop? These questions concerning organization and function have dominated research in the neurobiology of working memory.

Maintenance (Rehearsal) Versus Manipulation (Cognitive Control)

The manipulation-versus-maintenance distinction suggested by Baddeley's model has been explored extensively by Michael Petrides and colleagues, who have concluded that the dorsal

and ventral regions of the prefrontal cortex perform qualitatively different processes ([Owen, Evans, & Petrides, 1996](#); [Petrides, 1994, 1996](#)). Their findings, summarized in [Figure 9.17](#), indicate that the ventrolateral prefrontal cortex (VLPFC) supports the active controlled encoding and retrieval of information. This ventrolateral prefrontal region, in interaction with posterior cortical regions, may contribute to the roles of Baddeley's proposed visuospatial sketchpad and phonological rehearsal loop (which seem indeed to be two separate entities, as we observe later, in the Visuospatial and Phonological–Verbal Working Memory section). In contrast, the DLPFC supports higher-order cognitive-control functions, such as monitoring and manipulation of stored information, functioning much like Baddeley's central executive.

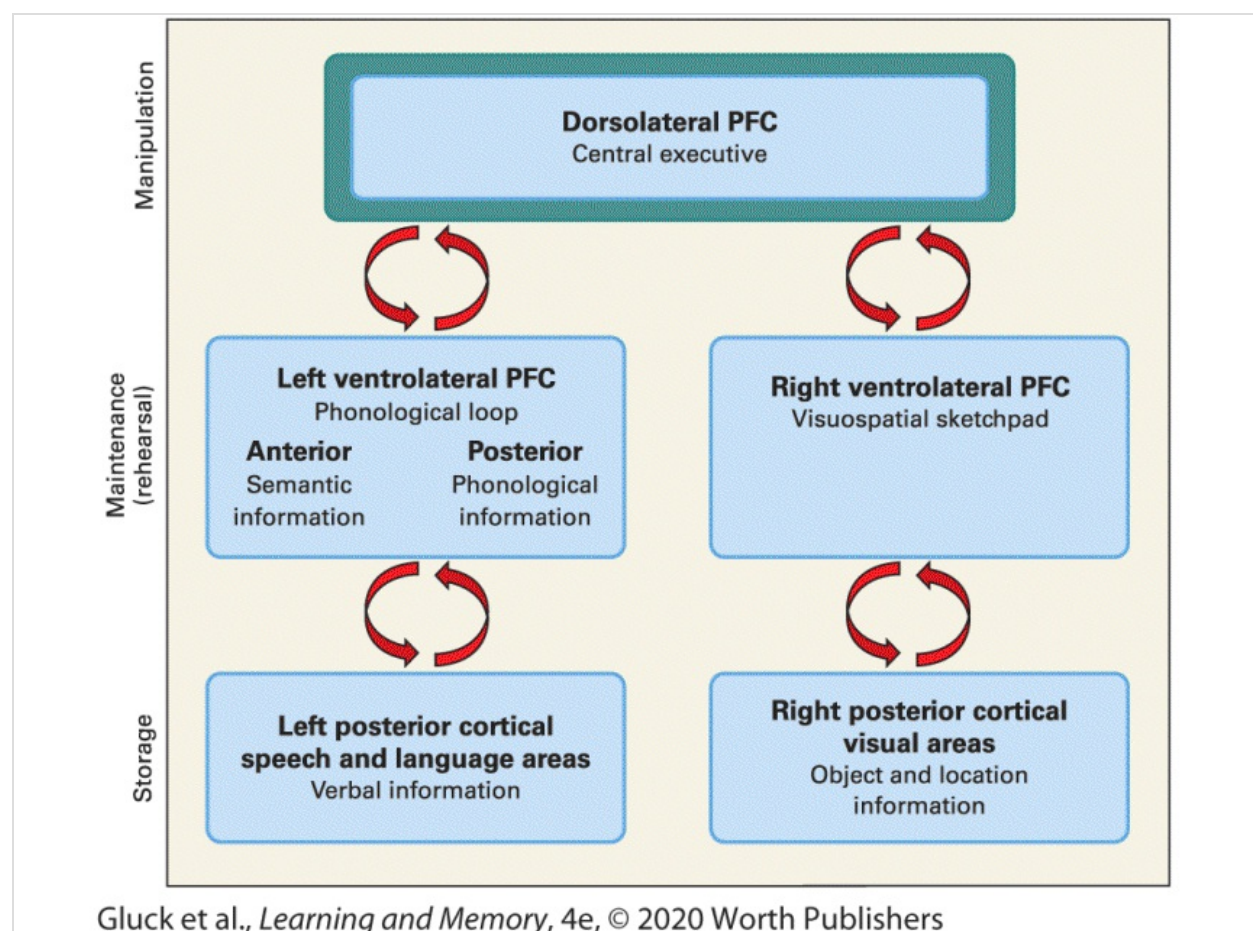


FIGURE 9.17 Brain substrates of working memory The dorsolateral prefrontal cortex supports higher-order cognitive-control functions, such as monitoring and manipulating of stored information, and acts much like Baddeley's central executive. The ventrolateral prefrontal cortex supports encoding and retrieval of information, performing the functions of the visuospatial sketchpad (right) and phonological rehearsal loops (left). Other brain regions named at bottom are connected to the VLPFC for maintenance of verbal and object and location information.

Description

The dorsolateral P F C termed as the central executive is linked by two directional arrows to a left ventrolateral P F C (phonological loop) and a right ventrolateral P F C (visuospatial sketchpad). The left ventrolateral P F C is divided into two parts as follows. Anterior-semantic information and Posterior-phonological information. The left ventrolateral P F C is linked to left posterior cortical speech and information areas (verbal information), and right ventrolateral P F C is linked to right posterior cortical visual areas (object and location information) by two directional arrows.

The dorsolateral P F C is responsible for manipulation, left ventrolateral P F C and the right ventrolateral P F C are responsible for maintenance (rehearsal), the left posterior cortical speech and language areas and the right posterior cortical visual areas are responsible for storage.

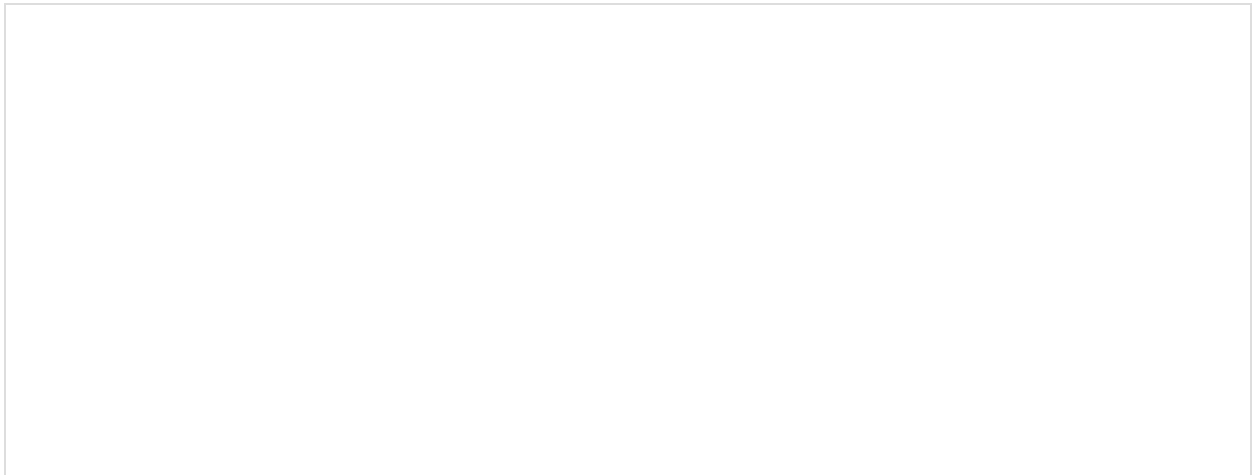
To test this mapping of processes to brain regions as portrayed in [Figure 9.17](#), Petrides and colleagues developed the self-ordered delayed-response tasks described in [Section 9.1](#). You'll recall that in the monkey version of this task (see [Figure 9.7](#)), the monkey obtains the most treats by remembering which of three containers it has already chosen (a memory manipulation). A 10-second delay, during which the containers are hidden, occurs between each opportunity to choose. Monkeys with lesions to parts of the DLPFC were severely impaired at this task and could not determine which containers had already been emptied and which still contained a reward. The spatial component of the information was made irrelevant in this task because the containers were moved randomly during the delays. By contrast, in another condition of the study, these same DLPFC-lesioned monkeys showed no problems solving basic delayed-*recognition* tasks; in other words, they were able to *maintain* object memories (i.e., simply recognize the objects) over varying delays, even though they had shown impairment in *manipulating* those memories ([Petrides, 1995](#)).

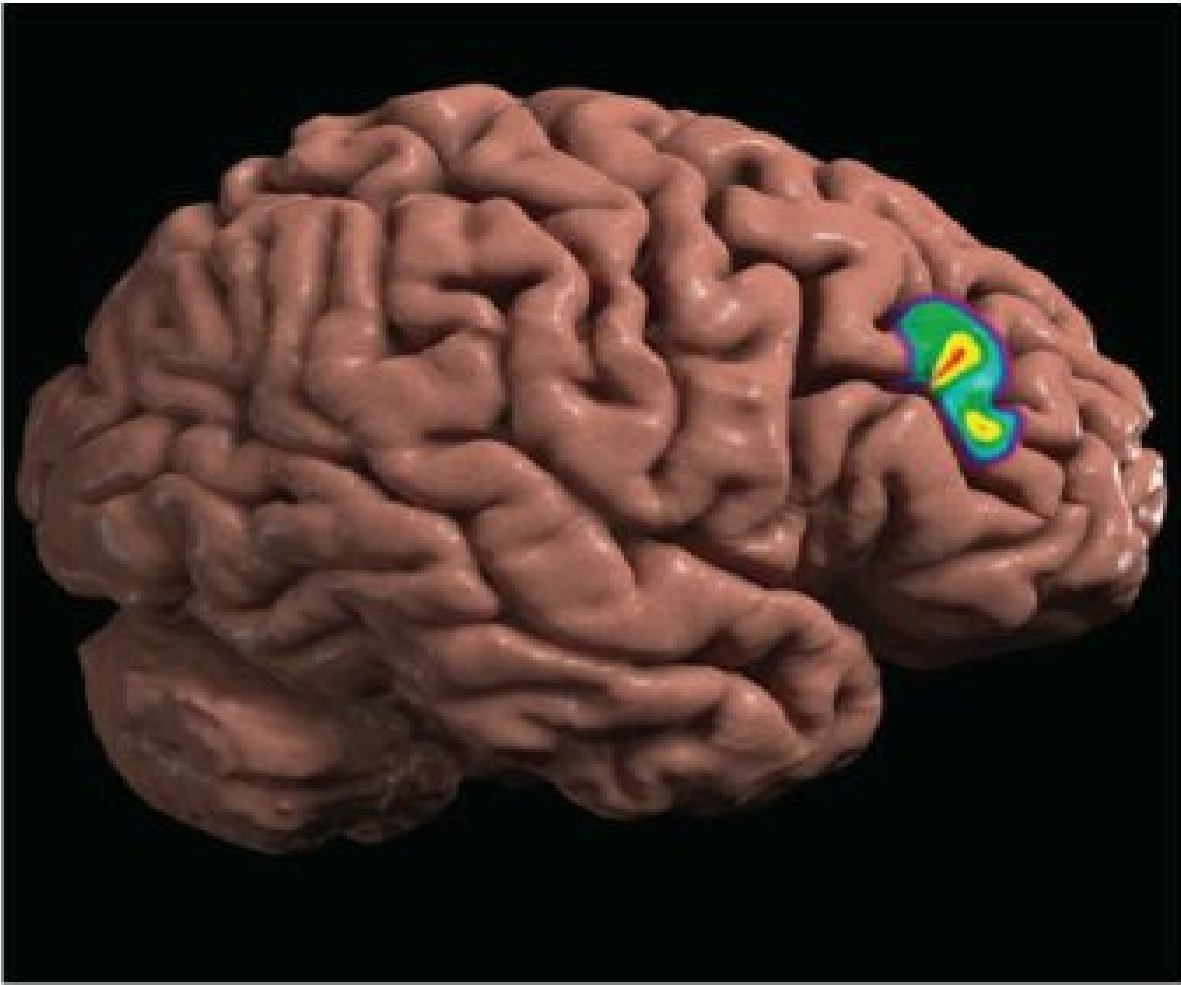
Later, [Petrides \(2000\)](#) showed that increasing the number of items to be monitored in these tasks exacerbated the impairments due to DLPFC lesions, whereas simply extending the delay time did not. Again, this finding implicates the DLPFC in monitoring (i.e., actively keeping tabs on and updating) but not in maintaining information in working memory. Thus, basic mnemonic judgments about recognition of recently seen objects, which require only maintenance of information during a delay, are not affected by DLPFC lesions. These maintenance functions are instead thought to be controlled by loops between the frontal lobes and more posterior regions of the brain, such as the posterior cortical speech and language areas (for verbal information) and the posterior cortical visual areas (for object and location information), as shown in [Figure 9.17](#).

Visuospatial and Phonological–Verbal Working Memory

As you learned in [Section 9.1](#), Baddeley’s model of working memory assumed the existence of two main memory buffers, one for visuospatial memory and the other for phonological–verbal memory. Studies of working memory in monkeys have, of course, been limited to studies of visuospatial memory because of the lack of verbal language in these nonhuman primates. All studies of phonological and verbal working memory have thus relied on the use of human participants. Despite such limitations, there is evidence to support the idea that visuospatial working memory and phonological–verbal working memory are produced in different parts of the brain. Studies of patients with selective brain damage to different parts of their frontal lobe suggest that the neural substrates of verbal working memory are localized more on the left side of the brain, while spatial working memory is more dependent on the right side of the brain.

Consistent with this finding of right-side dominance in visuospatial working memory from patient studies, Petrides and colleagues have conducted functional brain-imaging studies of healthy individuals performing self-ordered tasks ([Petrides, Alivisatos, Evans, & Meyer, 1993](#)). They found that when the items to be monitored were abstract designs (i.e., were visuospatial in nature), the self-ordered tasks produced significant activity in the DLPFC, predominantly in the right hemisphere ([Figure 9.18](#)). In contrast, when the items consisted of verbal material, the activity was more intense in the left hemisphere, although strong activity occurred in both the left and right sides of the DLPFC. From these results, the researchers concluded that while the right DLPFC has a dominant role in all monitoring processes, the left DLPFC is specialized for verbal materials.





Posterior
(back)



Anterior
(front)

Image courtesy of
Dr. Arthur W Toga,
Laboratory of Neuro
Imaging, University of
Southern California.

FIGURE 9.18 Brain imaging during self-ordered tasks Imaging data from a self-ordered task in which subjects had to remember previous selections made from a set of abstract designs show predominantly right-hemisphere activity in the prefrontal cortex.

Behavioral studies reviewed earlier in this chapter have indicated that verbal working memory retains items in a phonological code based on the sounds of the words and that these items are maintained through a rehearsal process similar to internally rehearsing speech ([Baddeley, 1986](#)). Consistent with the general tendency for language to be left-lateralized in the brain, frontal-lobe patients with damage to the left side are most likely to show specialized deficits in verbal (as opposed to visuospatial) working memory ([Shallice, 1988](#)).

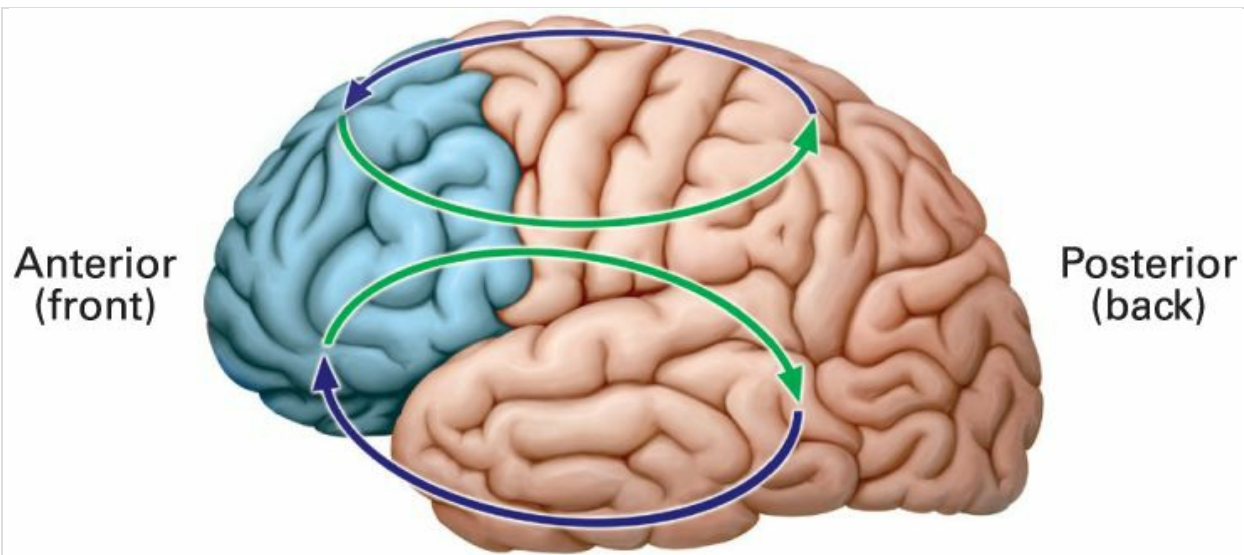
The Neural Bases of State-Based Accounts of Working Memory

Previously, we discussed early work by Fuster and Goldman-Rakic that demonstrated the importance of the frontal cortex for working memory ([Funahashi et al., 1989](#); [Fuster, 1973](#)). The sustained activity seen in the frontal cortex during delay periods in working memory tasks was originally interpreted as suggesting that this region is the site of temporary storage of items in working memory—in essence, a place-model interpretation. However, increasing evidence (much of it reviewed earlier in this chapter) suggests that the frontal-cortex activations reflect not the *storage* of information in working memory but rather the executive *processes* that are needed to maintain the representations of memory items in posterior areas of cortex, where they are permanently stored ([Postle, 2006](#)).

Computational modeling and lesion data alike have argued that both working memory and long-term memory (LTM) representations are stored in the same posterior cortical regions involved in the initial perception, encoding, and long-term storage of memories ([Jonides et al., 2008](#)). Data from human brain-imaging studies have also argued in favor of a common storage for both LTM and working memory in posterior brain regions ([Nee & Jonides, 2013](#)). For example, the patterns of activity seen in the posterior cortices when participants are making judgments about information recalled from LTM (e.g., the likability of famous people or the desirability of visiting famous locations) are similar to the patterns seen when the same participants encounter the same entities or objects in a working memory task that involves delayed recognition of pairs of items ([Lewis-Peacock & Postle, 2008](#)).

It is well established that posterior cortices are specialized for different sensory modalities (e.g., visual cortex, auditory cortex). In fact, this specialization of different regions, with their modality-specific limits, suggests a way of integrating Baddeley's distinct visuospatial and phonological loops (see [Figure 9.3](#)) into the state-based unitary model of memory.

The newer state-based approach to theories of working memory views working memory as a property of many different brain systems, including those that underlie semantic and episodic memory, sensory processing, and motor control ([D'Esposito & Postle, 2015](#)). How is working memory created in the brain through the operation of these various systems? One way is through persistent neural activity—much as seen in previously described studies of the frontal cortex. Other studies have shown that changes in synaptic properties in posterior cortical systems may also contribute to altering the state of those systems so as to produce the active attention that is the nature of working memory ([Erickson, Maramba, & Lisman, 2010](#)). Through such changes, networks of neurons located anywhere in the brain—from primary sensory cortices to multimodal association cortices—can activate stored information in a temporary fashion, allowing it to be used as working memory to support a variety of goal-directed behaviors ([D'Esposito & Postle, 2015](#)). Thus, working memory emerges from a network of brain regions, all of which send and receive information to and from the prefrontal cortex. Together, these networks accomplish the active maintenance of internal representations necessary for goal-directed behavior ([D'Esposito, 2007](#); [Postle, 2006](#)), as conceptualized in [Figure 9.19](#).



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

FIGURE 9.19 Maintenance in working memory through frontal–posterior circuits This schematic representation of frontal–posterior connections illustrates two sample loops, one dorsal and the other ventral. For each, the frontal activity projects back to the posterior areas (along green arrows), which in turn project forward to the frontal lobes (along blue arrows). The net result is that frontal activity induces sustained activation of posterior regions.

Description

Two looped circuits are shown on the dorsal and the ventral sides of the brain, linking the anterior and the posterior parts of the brain. Each loop is made of an arrow pointing from the anterior to the posterior of the brain and vice versa.

Goal Abstraction and Frontal-Lobe Organization

“What are you doing?” asks your roommate. Any of several answers would be equally correct: “Making a sandwich,” “Spreading peanut butter on bread,” or “Moving this knife from left to right.” These three answers all accurately describe what you are doing at the moment, but they do so at different levels of abstraction. “Making a sandwich” is the most abstract description of your current activity, and it also describes your ultimate goal. There are, of course, many different ways to make a sandwich, as well as a wide variety of ingredients. “Spreading peanut butter on bread” is thus a more specific description of what you are doing and also defines a more specific goal: it begins to identify what type of sandwich you are making. To spread the peanut butter on the bread, however, requires a specific action: the movement of the knife across the bread from left to right (and then back, from right to left).

Keeping goals of various degrees of abstraction in working memory as you proceed with a task has long been known to involve the frontal lobes. This is why people like Penfield's sister, who had frontal-lobe damage, have so much trouble cooking (including, most likely, preparing peanut butter sandwiches). The Tower of Hanoi puzzle (see [Figure 9.8](#)) also requires keeping a wide range of goals in mind, from the most abstract level ("Move all disks to the right-hand peg") to the most specific ("Move the yellow disk to the middle peg"). Impairments in solving the Tower of Hanoi have for many years been recognized as a sign of frontal-lobe dysfunction.

Although neuroscientists have long known that keeping goals in working memory while working on complex tasks depends heavily on the frontal lobes, recent research has suggested that the gradient of abstraction from general plans and goals to more specific action plans follows a physical gradient beginning at the front of the frontal lobes and moving back ([Badre, 2008](#); [Badre & D'Esposito, 2007](#)). As illustrated in [Figure 9.20](#), the most abstract plans (make sandwich; move all disks to the right) depend on the most anterior (frontmost) part of the frontal lobes. If the goals and plans to be maintained in working memory are specific and concrete (e.g., spreading peanut butter on the sandwich), they are likely to be localized in the more posterior regions of the frontal lobes.

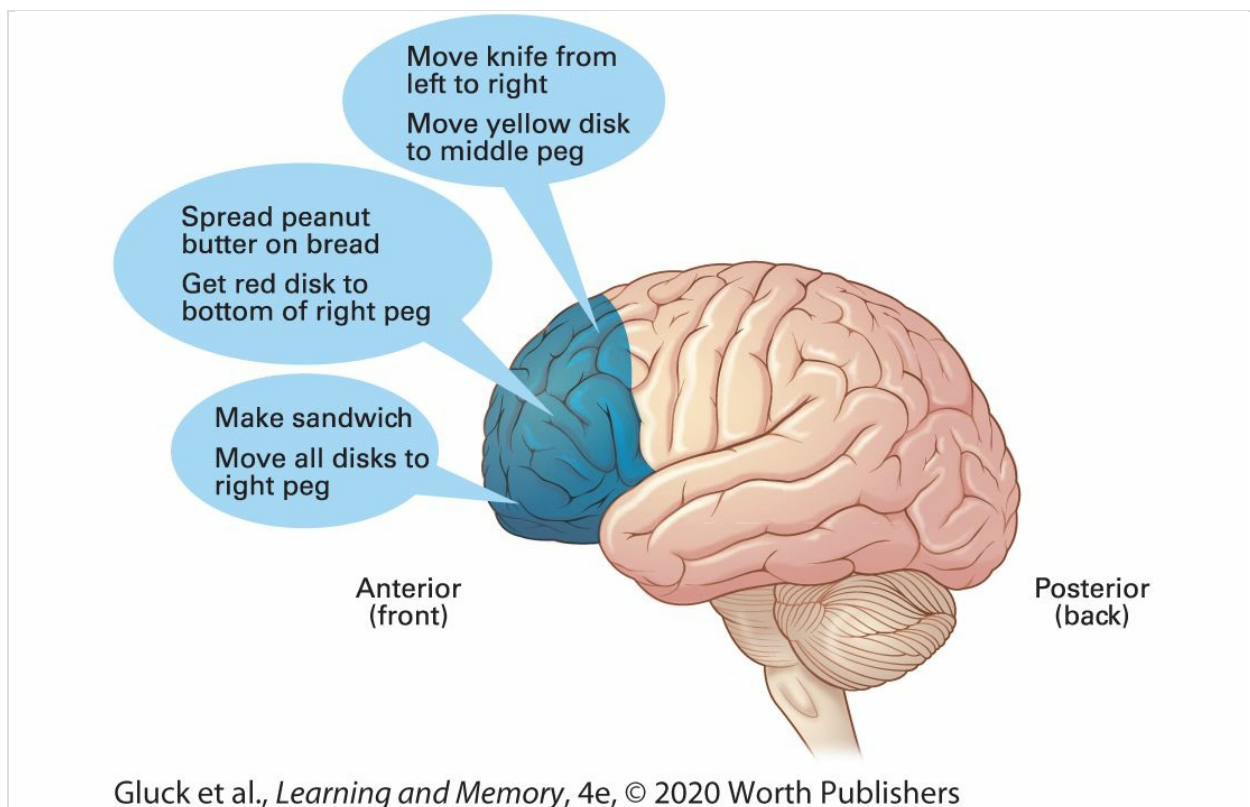


FIGURE 9.20 The anterior–posterior gradient of goal abstraction within the frontal cortex Three levels of abstraction of goals are shown for two examples—the making of a sandwich (top text in each balloon) and the Tower of Hanoi task (lower text)—illustrating approximate locations in the frontal cortex for control of each level of abstraction.

Description

Three callouts pointing to three different spots on the prefrontal cortex read as follows. 1-Move knife from left to right, Move yellow disk to middle peg. 2-Spread peanut butter on bread, Get red disk to bottom of right peg. 3-Make sandwich, move all disks to right peg.

Some of the evidence for this gradient of abstraction comes from people with brain damage due to stroke: people with damage toward the front of their frontal lobes are most impaired at tasks that require cognitive control at a high level of abstraction. Converging data come from lesion studies in monkeys that show a difference between the effects of posterior frontal-lobe lesions and the effects of mid-frontal-lobe lesions. Lesions to the more posterior regions of the frontal lobes disrupt performance in domain-specific motor learning tasks but not in domain-general monitoring; in contrast, lesions to the middle part of the DLPFC impair performance in general monitoring tasks but not in domain-specific tasks ([Petrides, 2006](#)).

Human functional imaging studies also provide converging evidence for this abstraction-specific gradient: tasks that require people to maintain more abstract, high-level goals activate a wider portion of the frontal lobes, including the more anterior regions toward the frontal pole of the brain ([Badre & D’Esposito, 2007](#); [Koechlin, Ody, & Kouneiher, 2003](#)). Imaging studies have also suggested that the flow of control within the frontal cortex goes from the most anterior regions (for the highest levels of abstract goals) toward the back of the frontal lobes (for the more specific subgoals), consistent with the idea that it is the desire to make a sandwich that drives you to butter the bread, not vice versa ([Koechlin et al., 2003](#)).

What if it was not your roommate who asked what you were doing but your kid brother? And what if he wanted to help? If your kid brother is 15, you might point him to the cabinets and say, “Make a sandwich for yourself.” But if he is only 6, you might hand him a piece of bread with peanut butter on it along with a (plastic) knife and instruct him to spread it back and forth. In other words, the older the child, the more likely you are to assign him a higher-level abstract goal. Younger children need more specific, concrete goals and plans. Might the difference in their abilities be due to differences in the maturity of the circuits in the more anterior parts of

their frontal lobes? Indeed, longitudinal data show that maturation of the circuit wiring in the frontal lobes progresses throughout childhood from the more posterior regions that support concrete plans toward the anterior regions that support more abstract planning, with the latter not maturing fully until late adolescence ([Shaw et al., 2008](#)). [Chapter 12](#) provides more information about critical periods in brain maturation and their impact on cognition across the lifespan.

Prefrontal Control of Long-Term Declarative Memory

What was the last movie you saw? To answer this question, you may have to perform a number of mental operations making use of your long-term memory. For example, you might search your memory by calling to mind every movie that you know to be currently playing, noting which ones you have seen and then trying to recall which of them you saw most recently. Alternatively, you could search your memory by thinking back over your recent activities in reverse chronological order. Knowing that you go out to movies on weekends, you might first think back to last weekend. Did you see a movie? If not, think back to the previous weekend. However you choose to set about answering this question, the process of searching your memory requires considerable strategic manipulation and control of memory processes as well as maintaining, throughout this search, an awareness of your ultimate goal: the name of the last movie you saw. This is exactly the kind of task that uses the prefrontal cortex in multiple capacities. Some patients with prefrontal-cortex damage exhibit significant deficits in retrieval of long-term memories ([Mangels, Gershberg, Shimamura, & Knight, 1996](#); [Shimamura, Jurica, Mangels, Gershberg, & Knight, 1995](#)).

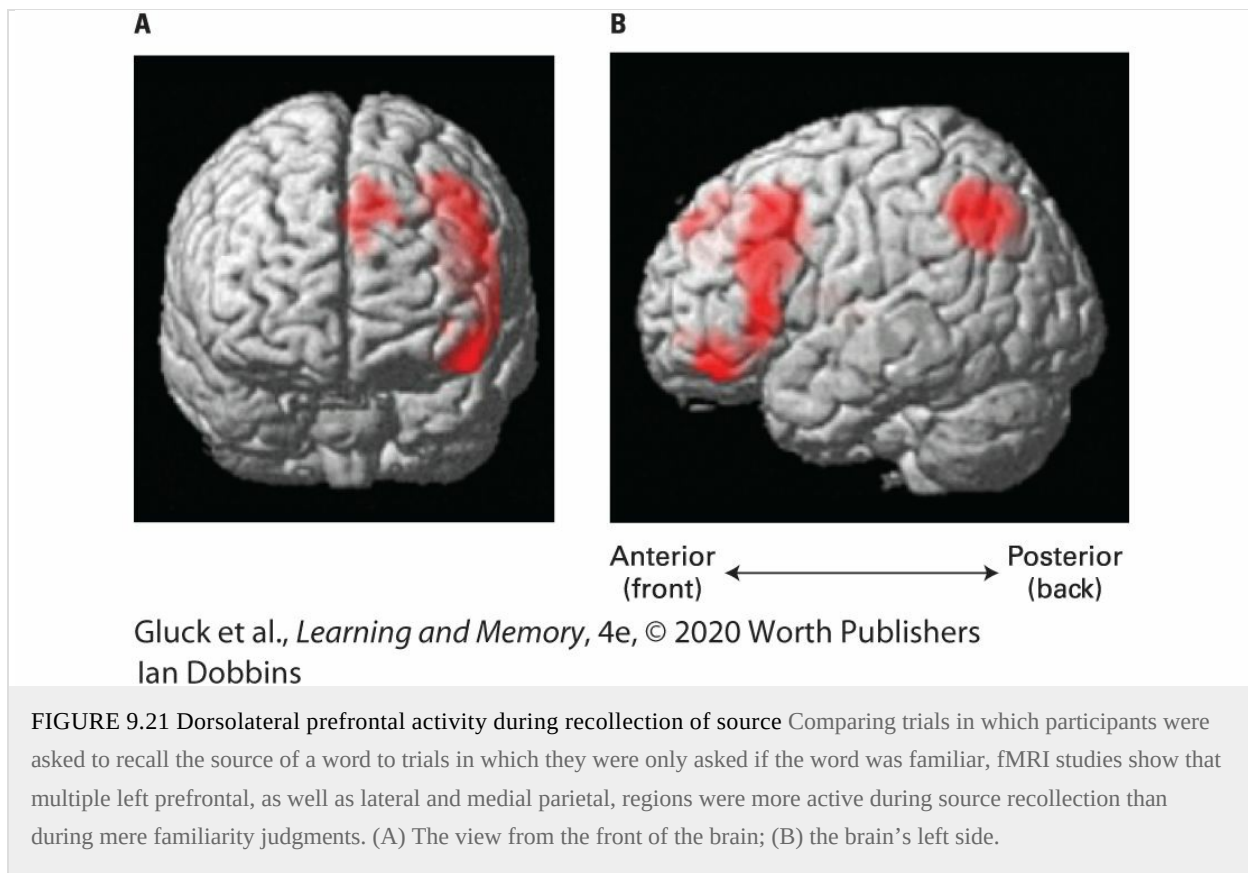
At the beginning of this chapter, we defined short-term memory as an active, temporary representation of information that either was just perceived or was just retrieved from long-term memory. Most of the discussion in this chapter has focused on the former class of information—information that was recently experienced. In what remains of this section, we briefly examine how working memory (and hence short-term memory) interacts with long-term memory. We focus especially on long-term memories of previously stored episodes or facts.

Neuroimaging has been particularly useful in furthering our understanding of how the frontal lobes and working memory guide the controlled search of long-term memory. Recall that

Petrides and colleagues argued that the ventrolateral prefrontal cortex supports active rehearsal and maintenance functions, whereas the dorsolateral prefrontal cortex supports higher-order executive-control functions, such as monitoring and manipulation of stored information ([Petrides, 2002](#)). Thus, the kinds of executive control and manipulation of memory needed for retrieval of specific episodic memories, such as the last movie you saw, should be subserved by the dorsolateral prefrontal cortex. In fact, this is exactly what functional neuroimaging has shown: the dorsolateral prefrontal cortex is activated during people's attempts to remember past events ([Nyberg, Cabeza, & Tulving, 1996](#); [Wagner, Desmond, Glover, & Gabrieli, 1998](#)).

Have you ever met someone at a party who seems familiar, but you can't remember how you know her? (Is she an elementary school classmate, or did you meet on that summer trip to Israel?) You just can't recall, but you do know you met before. On the other hand, very often you will see a person and not only realize that he is familiar but immediately remember how and where you met. (The former phenomenon, known as the tip-of-the-tongue phenomenon, is introduced in [Chapter 7](#), where it is identified as the feeling of trying to recall information that is not forgotten but merely temporarily inaccessible.)

According to a study by Anthony Wagner, Daniel Schacter, and colleagues, you probably used your dorsolateral prefrontal cortex in the latter situation, in which you recollected the source of your memory, but not in the former situation, in which you knew that the person was familiar but could not remember why ([Dobbins, Foley, Schacter, & Wagner, 2002](#)). In their study, people were shown various words and asked one of two questions: "Is it abstract or concrete?" or "Is it pleasant or unpleasant?" Later, the participants were shown the words again and were asked either whether they remembered seeing the word during the first part of the experiment (i.e., did they recall whether the word was considered at all?) or whether they remembered which task the word appeared in (i.e., did they recall whether they judged it on the concrete/abstract dimension or on the pleasant/unpleasant dimension?). As shown in [Figure 9.21](#), the dorsolateral prefrontal cortex was more active when people were asked to recall the source of the word (i.e., which task it was used in) than when they were asked whether or not the word had appeared at all (regardless of task).



Description

The view from the front of the brain in section A highlights two areas on the left side of the brain. The view from the left side of the brain highlights two areas in the anterior part and a smaller area in the posterior part. A double-headed arrow points toward the posterior and the anterior parts of the brain.

If, as Petrides argued, the ventrolateral prefrontal cortex supports active rehearsal and maintenance functions, then we might expect to see more VLPFC activity during intentional active encoding when participants are explicitly told to remember something, in contrast to the DLPFC activity seen in retrieval ([Petrides, 2002](#)). Functional imaging studies using fMRI and PET have indeed reliably shown that intentional encoding of new memories activates the VLPFC. Because most of these studies used meaningful stimuli, such as images of nameable real-word objects, the *left* VLPFC was primarily activated, consistent with the general tendency of the left prefrontal cortex to be specialized for verbal processing ([Nyberg et al., 1996](#)).

The functional role of the left VLPFC during encoding of new semantic information can be further subdivided into the contributions of its anterior (front) and posterior (back) regions, as

illustrated in [Figure 9.17](#). Anterior regions are activated during tasks that involve semantic processing ([Thompson-Schill, D’Esposito, Aguirre, & Farah, 1997](#)), while posterior regions are activated during phonological processing ([Buckner, Raichle, Miezin, & Petersen, 1996](#)). Thus, remembering the name of a wealthy new acquaintance, Bill, by noting that he probably has lots of bills in his wallet (a *semantic* elaboration of a meaning of the word “bill”) would likely involve processing by your *anterior* ventrolateral prefrontal cortex. In contrast, rehearsing a complex foreign-sounding name over and over likely involves *phonological* processing in the *posterior* ventrolateral prefrontal cortex.

Further support for this anterior–posterior differentiation comes from a study that compared brain activity of people making either a semantic analysis of words (“Is it abstract or concrete?”) or a phonological analysis (“How many syllables does it contain?”). Although the posterior region of the left ventrolateral prefrontal cortex was activated during both tasks—reflecting a common phonological component—only the semantic task resulted in activation of the anterior left ventrolateral prefrontal cortex ([Poldrack, Prabhakaran, Seger, & Gabrieli, 1999](#)). In contrast, other researchers subsequently demonstrated that nonsemantic tasks that involved only phonological processing activated the posterior, but not the anterior, regions of the left ventrolateral prefrontal cortex ([Wagner, Koutstaal, Maril, Schacter, & Buckner, 2000](#)). Refer again to [Figure 9.17](#) for a schematic map of which type of working memory tasks involve which brain regions.

Overall, there are numerous parallels between the role of the prefrontal cortex in working memory (along with the precise location of its activity) and its role in episodic memory. Thus, the control processes and rehearsal mechanisms implicated in working memory appear to also play crucial roles in the encoding and retrieval of long-term memories for episodic and semantic information ([Wagner, 2002](#)).

TEST YOUR KNOWLEDGE

Functional Neuroanatomy of the Prefrontal Cortex

Identify the region of the prefrontal cortex whose activity is most critical for each of the following tasks. ([Answers appear at the end of the chapter.](#))

1. Deciding who should sit where around a dinner table set for eight to avoid seating ex-spouses and feuding ex-business partners next to each other
2. Rehearsing the toast you will make at your brother’s wedding

3. Learning the definitions of “distributor,” “ignition coil,” and “carburetor” by reading a car-repair manual
4. Remembering how to pronounce the name of the French exchange student you just met
5. Remembering where you parked and deciding which way to walk to your parking spot as you exit the department store at the mall

Interim Summary

- Studies with animals as well as humans identify the frontal lobes of the brain—especially the prefrontal cortex (PFC), the most anterior section of the frontal lobes—as being critical for working memory and cognitive control.
- The ventrolateral prefrontal cortex (VLPFC) supports encoding and retrieval of information (including rehearsal for maintenance), performing as the visuospatial sketchpad and phonological rehearsal loop proposed by Baddeley; the dorsolateral prefrontal cortex (DLPFC) supports higher-order executive-control functions such as monitoring and manipulating of stored information, thus doing the job of Baddeley’s central executive.
- Working memory is now believed to result from functional interactions between the prefrontal cortex and the rest of the brain.
- The more concrete and specific the goals or plans that must be maintained in working memory, the more posterior the localization of their function within the frontal lobes, forming an anterior-to-posterior gradient from abstract to specific.
- During encoding of new verbal information, the *anterior* prefrontal cortex is activated for tasks that involve *semantic* processing, while the *posterior* prefrontal cortex is activated for *phonological* processing.

9.3 Clinical Perspectives

Research on the role of the prefrontal cortex in working memory and cognitive control has provided clues for improving the diagnosis and treatment of several common neurological and psychiatric disorders. Two of the most common disorders involving dysfunctional prefrontal circuits are schizophrenia and attention-deficit/hyperactivity disorder (ADHD).

The Prefrontal Cortex in Schizophrenia

[Chapter 6](#) describes some of the memory deficits associated with hippocampal dysfunction in schizophrenia. In the current chapter, our discussion of schizophrenia focuses instead on the ways in which frontal-lobe dysfunction contributes to symptoms of the disorder. People diagnosed with schizophrenia display disturbances in both cognition and memory, especially in working memory and executive control ([Forbes, Carrick, McIntosh, & Lawrie, 2009](#)). This finding is consistent with a wide range of other data suggesting that the dorsolateral prefrontal cortex is dysfunctional in schizophrenia. In contrast, functions attributed to the ventrolateral prefrontal cortex seem relatively unimpaired in patients with schizophrenia. For example, people with schizophrenia demonstrate close to normal performance on phonological or visuospatial memory tasks ([Barch, Csernansky, Conturo, Snyder, & Ollinger, 2002](#)) and on memory tasks involving only minimal delays or few items to keep track of ([Park & Holzman, 1992](#)). However, patients with schizophrenia are especially impaired at visuospatial working-memory tasks when these tasks involve the manipulation or updating of information in working memory ([Park & Holzman, 1992](#)). Similar cognitive-control deficits are also seen in close relatives of schizophrenia patients ([Park, Holzman, & Goldman-Rakic, 1992](#)).

Neuroimaging provides further insights into prefrontal-cortex dysfunction in schizophrenia. Daniel Weinberger and colleagues presented the first neuroimaging evidence for dorsolateral prefrontal-cortex dysfunction in schizophrenia by measuring blood flow in different cerebral regions ([Weinberger, Berman, & Zec, 1986](#)). They found that when patients with schizophrenia tried to solve the Wisconsin Card Sorting Test (see [Figure 9.9](#)), their dorsolateral prefrontal cortex showed no evidence of increased blood flow as it would in control individuals. Moreover, there was a correlation among the schizophrenia subjects between the amount of blood flow in this region and performance: the greater the blood flow in the dorsolateral prefrontal cortex, the better the patients performed on the Wisconsin Card Sorting Test.

More recent studies provide further evidence for localization of a cognitive-control deficit in schizophrenia within the dorsolateral prefrontal cortex. For example, researchers found that schizophrenia correlates with depressed dorsolateral prefrontal-cortex activity during the *N*-back task, which, as you learned in [Section 9.1](#), is a standard test of the manipulation of working memory. Ventral and posterior prefrontal-cortex activity, however, is normal in these patients, suggesting that rehearsal mechanisms, associated with these ventral and posterior areas, are less affected by schizophrenia ([Barch et al., 2002](#)). These neuroimaging results are consistent with postmortem studies of schizophrenia patients that reveal neural pathologies in the dorsolateral prefrontal cortex but not in more ventral regions.

Inefficient Prefrontal Cortical Systems in Schizophrenia

Recent functional brain-imaging studies of patients diagnosed with schizophrenia suggest that their reduced capacity for executive aspects of working memory is accompanied by engagement of a larger network of other cortical regions. This is consistent with the idea that they are recruiting compensatory networks that utilize other, less impaired brain regions ([Tan, Callicott, & Weinberger, 2007](#)). Even when patients are able to keep up with the processing demands of tasks that require cognitive control and do exhibit normal working-memory behaviors, brain imaging shows that these patients are meeting the demands less efficiently by employing greater posterior cerebral metabolic brain activity with less focused cortical activity.

Recall from earlier in this chapter the data indicating that higher-order processes in the dorsolateral prefrontal cortex exert control over more ventrolateral regions responsible for less complex processes, such as rehearsal of information within working memory ([D'Esposito et al., 1995](#); [Koechlin et al., 2003](#)). In comparison, brain imaging in schizophrenia patients shows increased patterns of activity in their ventrolateral prefrontal cortex, even during tasks where the patients show near-normal working-memory performance ([Tan, Choo, Fones, & Chee, 2005](#); [Tan et al., 2006](#)). Evidently, while control subjects use the dorsolateral prefrontal cortex in executive working-memory tasks, schizophrenia patients are often unable to do so; rather, they engage greater ventrolateral prefrontal cortex involvement, possibly as a form of compensation for the dysfunctional dorsolateral prefrontal response.

Dopamine and the Genetics of Schizophrenia

What is wrong with the dorsolateral prefrontal cortex in schizophrenia patients? One view is that

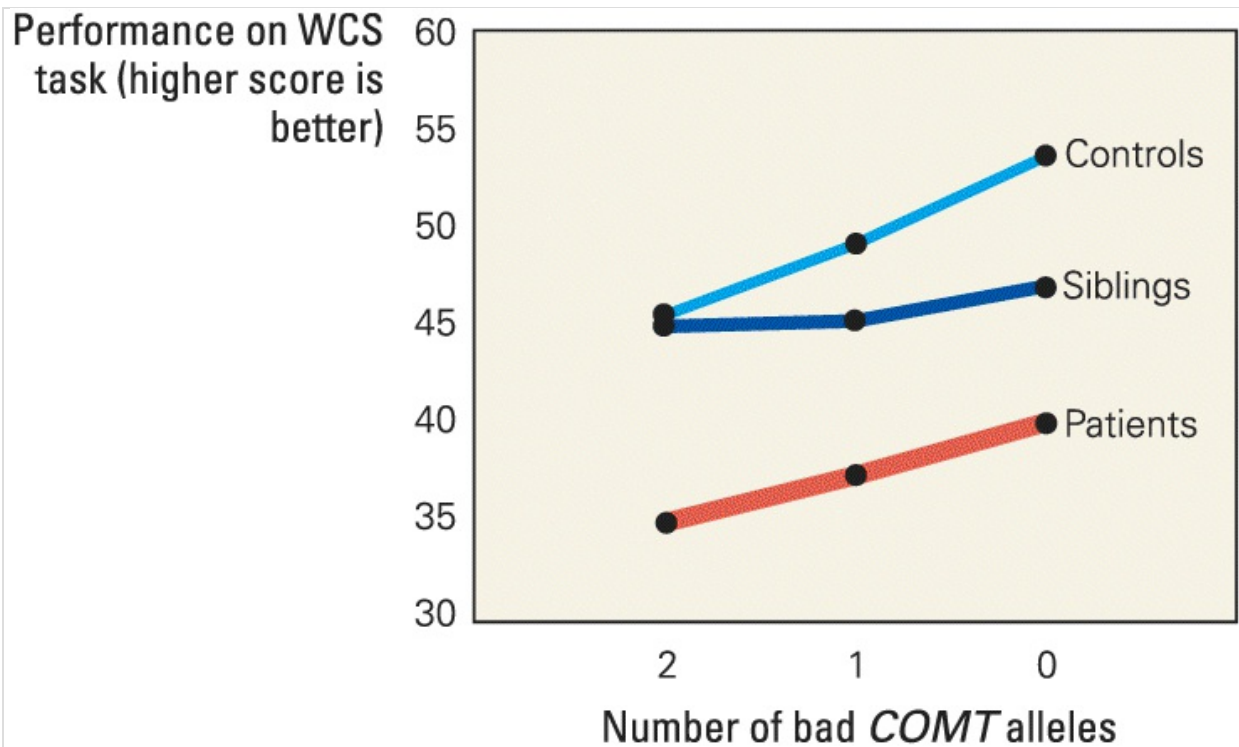
the deficits in working memory and executive control found in schizophrenia may be linked to deficiencies in cortical dopamine processing. [Dopamine](#) is a neuromodulator that alters neuron-to-neuron communication, and most pharmacological treatments for schizophrenia work by altering the transmission of dopamine.

dopamine

A neuromodulator that alters neuron-to-neuron communication.

Genetic research into the causes of schizophrenia includes a search for genes that convey a heightened susceptibility for the disease. For example, Daniel Weinberger and colleagues have shown that mutation in the *COMT* gene affects dopamine metabolism in the frontal lobes ([Egan et al., 2001](#)). *COMT* is one of several enzymes that are critical for degrading dopamine (and other related neuromodulators). Several pharmaceutical treatments target the *COMT* enzyme to alter its activity, thereby affecting the availability of dopamine and related neuromodulators. For example, *COMT* inhibitors prevent the breakdown of dopamine and are used as a treatment in Parkinson's disease to increase levels of dopamine in the brain.

In control subjects, the status of the *COMT* gene was seen to predict 4% of the variance in performance on the Wisconsin Card Sorting Test. As shown in [Figure 9.22](#), having 2, 1, or 0 copies of the less effective allele for the *COMT* gene predicted performance score on the Wisconsin Card Sorting Test. (Higher values on the graph indicate better performance.) Patients diagnosed with schizophrenia (bottom curve on graph) who had two copies of the less effective allele showed worse performance on this task than did patients with one or none of the bad *COMT* alleles. A similar pattern—but higher overall performance—was seen in both nonsymptomatic siblings of schizophrenia patients and nonsymptomatic controls drawn from the general population. Thus, in addition to the main between-group effects of controls doing better than siblings and siblings doing better than patients, there was also a within-group effect based on numbers of genes. For all three groups, the number of the bad *COMT* alleles affected performance. This finding suggests that a mutation in one kind of gene causes only a small change in cognitive performance but that a combination of mutations in many different genes could push a person past a tipping point into a high-risk category for schizophrenia.



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

FIGURE 9.22 Effect of a gene mutation on frontal-lobe function A higher number of copies of a less effective *COMT* allele correlates with poorer performance on the Wisconsin Card Sorting Test in schizophrenia patients, their siblings, and controls. (A higher score on the graph in this figure means better performance.)

Data from [Egan et al., 2001](#).

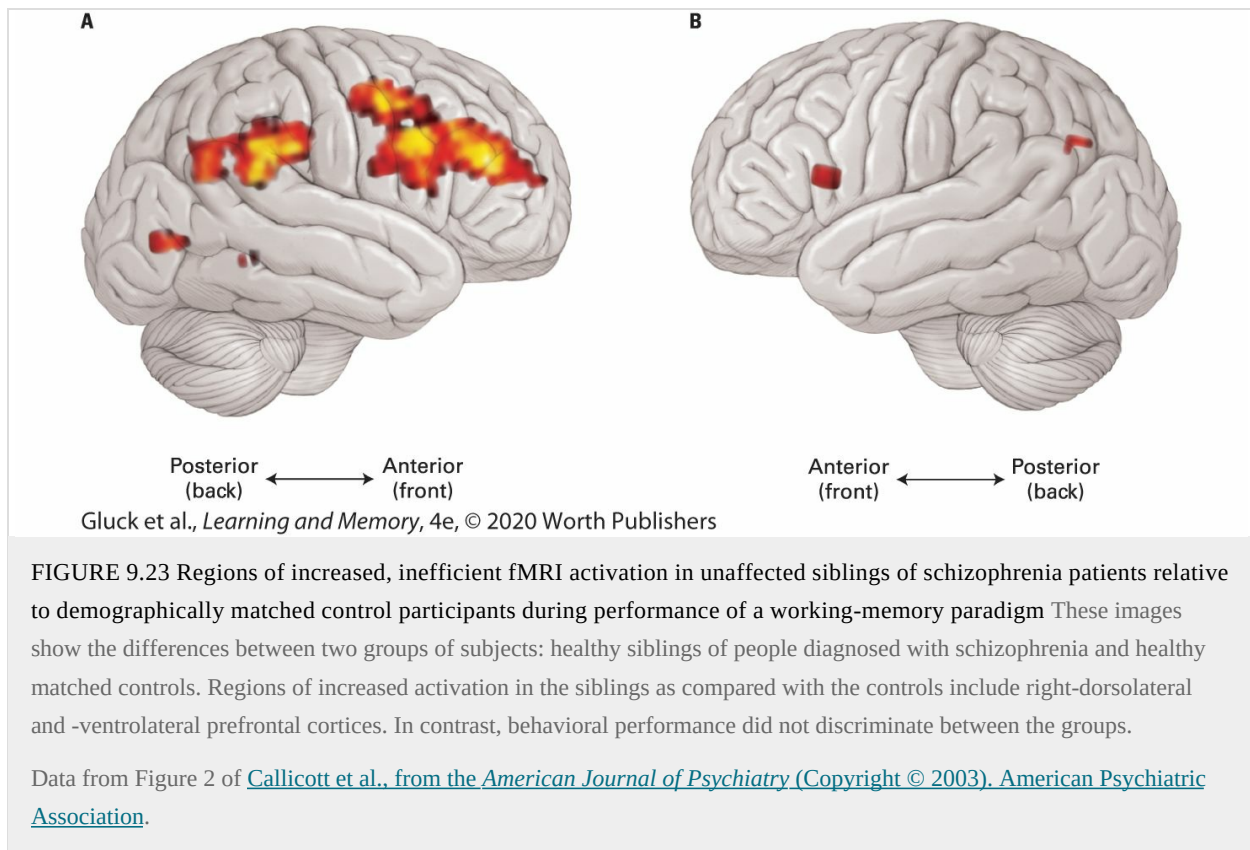
Description

The readings on the vertical axis range from 30 to 60 in increments of 5 and the readings on the horizontal axis range from 2 to 0 in decreasing units of 1. The graph shows three curves representing patients, siblings, and controls. Each curve runs through three marked points as follows. Patients: (2,35), (1,37), and (0,40). Siblings: (2,45), (1,45), and (0,47). Controls: (2,46), (1,48), and (0,55).

Weinberger and colleagues also used the 2-back task, with its heavy dependence on working memory and executive control, to do brain-imaging studies of the effects of the *COMT* gene. They looked at the brains of nonsymptomatic individuals with 0, 1, or 2 copies of the less effective *COMT* allele to see which brain regions showed activity during the 2-back task. The region that was most highly correlated with this allele was the prefrontal cortex. The more copies of the less effective *COMT* allele (and hence the worse the dopamine functioning), the less

prefrontal-cortex activity was seen during the 2-back task. This suggests that having 1 or 2 copies of the less effective allele (as is most common in those with schizophrenia) impairs activation of the prefrontal cortex during working-memory and executive-function tasks. These studies provide evidence that genetic mutations affecting dopamine activity in the prefrontal cortex are related to the emergence of the cognitive deficits seen in schizophrenia.

As noted earlier, increased prefrontal activation is thought to reflect inefficient function in the prefrontal areas, such that the increased activity is necessary to support a given level of performance. Several groups have now shown that this prefrontal inefficiency is also present in certain unaffected siblings of schizophrenia patients. The siblings show greater prefrontal activation than do controls, even though their task performance does not differ from that of matched controls ([Figure 9.23](#)). These results suggest that physiological evidence derived from neuroimaging may provide a more direct reflection of the effects of genes (e.g., on the level of dopamine metabolism) than do behavioral changes.



Description

The imagery in section A shows many areas in the anterior part, and few areas in the posterior part of the brain highlighted. The imagery in section B shows a small spot in the anterior part, and a very small spot in the posterior part highlighted. A double-headed arrow points toward the posterior and the anterior parts of the brain.

Attention-Deficit/Hyperactivity Disorder

Attention-deficit/hyperactivity disorder (ADHD) is one of the most commonly diagnosed psychiatric problems in children. About 5% of children are diagnosed with ADHD, although its true prevalence is hard to estimate, given that the diagnostic definition of ADHD is constantly being refined. Children and adults with this disorder have great difficulty with cognitive-control processes such as planning, organizing their time, keeping attention focused on a task, and inhibiting responses to distracting stimuli. ADHD symptoms are especially evident in settings that require the exercise of self-control. For example, children diagnosed with ADHD can sit still for hours when they are actively engaged in playing interactive video games, but they have trouble paying attention in school, where they must sit for long periods and pay attention to less captivating information. Given these problems, it is not surprising that students with ADHD are at elevated risk for academic underachievement ([Martinussen & Major, 2011](#)).

Most researchers and clinicians believe that ADHD involves dysfunction in the prefrontal cortex and its cortical and subcortical connections ([Solanto, Arnsten, & Castellanos, 2000](#)), including connections to the cerebellum and the basal ganglia. The fact that ADHD patients have difficulty sustaining a behavior or thought over a delay is consistent with the data, reviewed earlier in this chapter, showing that prefrontal cortical cells play a critical role in regulating thought and behavioral output through their sustained firing over delays in the absence of external input. Structural neuroimaging of children with ADHD shows that they have a smaller right prefrontal cortex, the region associated with spatial attention and working memory. Functional brain-imaging studies have shown evidence of decreased prefrontal activity in ADHD, while diffusion-tensor imaging has indicated weaker prefrontal white matter connections ([Arnsten, 2006](#); [Castellanos & Tannock, 2002](#)).

Behavioral research suggests that working memory in particular is impaired in patients with ADHD. People with ADHD show marked deficits in mental calculations that require use of working memory ([Schweitzer et al., 2000](#)). As with schizophrenia, current medications for ADHD act in part by altering dopamine function in the cortex. The most common treatments for

ADHD, such as Ritalin (a brand name of methylphenidate), are stimulants that either increase dopamine release or block its reuptake at synapses. Unfortunately, the effects of these medications are temporary, and the behavioral problems often reappear after 3 or 4 hours.

There is strong evidence for frontal-cortex dysfunction in ADHD, but is the frontal cortex the *origin* of the attentional and cognitive control problems of ADHD? Some investigators have suggested that frontal-cortex dysfunction, and the associated working-memory and attentional problems, are actually the consequence of deficits in other subcortical structures, especially the basal ganglia ([Volkow, Wang, Fowler, & Ding, 2005](#); [Volkow et al., 2004](#)).

The basal ganglia, as described in [Chapters 5](#) and [8](#), are critically involved in skill learning and action selection. But if the origins of ADHD are in the basal ganglia, not the frontal lobes, why do stimulants improve working memory and reduce symptoms of ADHD? One view, for which there is some neuroimaging evidence, is that people with ADHD have “noisy” basal ganglia that sometimes send inappropriate signals to the prefrontal cortex, resulting in distractible behavior, while at other times the basal ganglia do not signal the prefrontal cortex when appropriate, resulting in perseveration or inattention ([Volkow et al., 2005](#)).

Perhaps the reason stimulant medications remediate the attentional problems in people with ADHD is that stimulant medications, by increasing extracellular dopamine in the striatal region of the basal ganglia, increase the saliency of the basal ganglia signal to the prefrontal cortex ([Volkow et al., 2004, 2005](#)). This hypothesis is consistent with studies (reviewed in [Chapter 5](#)) that describe a key role for dopamine in the basal ganglia (as well as in the frontal lobes). Recent brain-imaging studies have argued that increased but inefficient activity in the prefrontal cortex, found during certain task conditions in children with ADHD, may serve a compensatory function for dealing with noisy input from the basal ganglia ([Sheridan, Hinshaw, & D’Esposito, 2010](#)).

Medications are not the only treatment for ADHD. Behavioral training methods may also help. Several studies have shown that training children with ADHD to better manage their cognitive control and to use their working memory more effectively can, over the course of several weeks, lead to improvements in their working memory on certain tasks, as well as reduce some of the core symptoms of inattention and hyperactivity ([Beck, Hanson, Puffenberger, Benninger, & Benninger, 2010](#); [Klingberg et al., 2005](#)). Thus, working-memory training appears promising as

an intervention for improving both cognitive functioning and core ADHD symptoms.

Like schizophrenia, ADHD is a heritable psychiatric disorder (which therefore tends to run in families), and scientists are hot on the trail of the genetic bases for this heritability. Recent research has identified some of the genes believed to be linked to ADHD. Like some of the genes associated with schizophrenia, these ADHD genes regulate the function of dopamine in the brain ([Durstun, 2010](#)). Future research will hopefully identify the genes more clearly, discover how they relate to the behavioral problems of ADHD, and thus provide clues for developing more effective treatments. Advances in diagnosis are also sorely needed: studying mental illnesses such as schizophrenia and ADHD is difficult because of the lack of clear disease markers, the lack of uniformly applied diagnostic criteria, and the growing realization that these disorders are highly heterogeneous, with each patient sharing some but not all symptoms with other patients in the same diagnostic category.

Interim Summary

- People with schizophrenia demonstrate working-memory impairments when attempting to maintain a large number of items in the mind over a temporal delay, an activity that requires functions associated with the dorsolateral prefrontal cortex. In contrast, functions attributed to the ventrolateral prefrontal cortex seem relatively unimpaired; thus, performance on phonological or visuospatial memory tasks and on memory tasks involving only minimal delays or few items appears normal.
- People with ADHD show deficits in mental calculations that require use of working memory.
- Neuroimaging of people with ADHD indicates that they have a smaller prefrontal cortex as well as noisy input signals from the basal ganglia that prevent the basal ganglia from accurately indicating what is or is not important to attend to.

Synthesis

Let's return for the last time to Kamila, to see what insights we have gained into the cognitive and other brain processes that she calls on throughout the day. Early that morning, a Tuesday, Kamila checks her schedule and homework assignments. Her dorsolateral prefrontal cortex is heavily activated while she sorts through her list, paying most attention to the things she must attend to first. This focusing of her attention helps her plan and organize for that particular day, triggering her memory to recall the items she will need to bring along with her. As she considers them, the various objects are briefly represented in her ventrolateral prefrontal cortex. As she then arranges the items in her backpack, the order and location of each one activate the spatial working-memory capabilities of her dorsolateral prefrontal cortex.

When she stops at the bank to get some cash, Kamila relies on her DLPFC to help retrieve her PIN from long-term memory, after which she rehearses the PIN through her phonological loop, activating her VLPFC. With cash in hand, she dashes straight to class, or would have done so had not her DLPFC been maintaining a reminder that tells her she has to switch from her normal routine, suppress her automatic tendency to go directly to business class, and instead make a side trip to the biology department to drop off her homework. She then has to remember that her business class will not be meeting in the usual location, once again requiring suppression of an automatic response.

Finally, Kamila arrives at business class. While she listens to the lecture, she also discretely reads bits and pieces of biology, relying on activity in her DLPFC to alert her whenever the speaker is discussing something new or especially important, at which times she tries to pay more attention to the lecture. What really grabs her attention, however, is his announcement of a surprise quiz. If Kamila had not been playing the piano and singing her own compositions at the cheerleading fundraiser party until 2 a.m., she might have had the time to review her reading in case of just such a quiz. Unfortunately, she didn't do any reviewing (although she had certainly been exercising her VLPFC recalling song lyrics). During the pop quiz, Kamila realizes she has far less knowledge in her long-term memory to draw on than she needs. She engages her DLPFC as she desperately tries to recall the answers to the quiz questions, but alas, she never learned the material in the first place.

All in all, it's a busy morning for Kamila as her prefrontal cortex, working closely with long-

term memory storage areas in the posterior parts of her brain, assists her in controlling her memory, mind, and life.

Chapter 9 Review

Know Your Key Terms

[central executive](#)

[cognitive control](#)

[delayed nonmatch-to-sample \(DNMS\) task](#)

[dopamine](#)

[dorsolateral prefrontal cortex \(DLPFC\)](#)

[dysexecutive syndrome](#)

[long-term memory \(LTM\)](#)

[perseveration](#)

[phonological loop](#)

[prefrontal cortex \(PFC\)](#)

[sensory memory](#)

[short-term memory \(STM\)](#)

[transient memory](#)

[ventrolateral prefrontal cortex \(VLPFC\)](#)

[visual sensory memory](#)

[visuospatial sketchpad](#)

[word-length effect](#)

[working memory](#)

Quiz Yourself

1. In the Tower of Hanoi puzzle, moving all the disks to the right is an abstract plan that depends on the _____ part of the frontal lobes, whereas specific plans for completing individual steps to solve the puzzle (e.g., moving the yellow disk to the middle peg) depend on a more _____ part of the frontal lobes. ([pp. 402–403](#))
2. In the Wisconsin Card Sorting Test, frequently used in clinical settings, patients with frontal-lobe lesions show _____; that is, they fail to learn a new rule despite feedback indicating that the old rule is incorrect. ([pp. 393–394](#))

3. It is hypothesized that in ADHD, projections to the frontal cortex from _____-containing neurons in the _____ are hypoactive or underdeveloped. ([p. 411](#))
4. Carol sustained frontal-lobe damage when she was hit by a car last year. Now she is unable to hold down a job, as she never cares to show up on time. Given her brain damage and behavior, it is likely that Carol has _____ syndrome. ([p. 393](#))
5. Jamie forgets the color of Carrie's shirt as soon as they exchange goodbyes. The fleeting visual sensation that Jamie had before she forgot the color is called a(n) _____. ([p. 371](#))
6. Jim can remember a long (seven- or eight-item) list of colors without much thought, but when asked to remember a list of full names, he can remember only four or five. From what we know about short-term memory storage, this is likely due to the _____. ([p. 376](#))
7. Schizophrenia patients with two less effective alleles of the *COMT* gene—a gene that functions in _____ metabolism—are more likely to perform _____ on the Wisconsin Card Sorting Task compared to patients with zero or one abnormal copy. ([pp. 408–409](#))
8. _____ is the active and temporary representation of information that is maintained for the short term in the mind, assisting the individual to think about and decide what to do next. ([p. 370](#))
9. The _____ in Baddeley's model monitors and manipulates both of the working-memory buffers, providing cognitive control of working memory. ([p. 375](#))
10. Many studies suggest that the _____ is dysfunctional in patients with schizophrenia. ([pp. 407–408](#))
11. Most pharmacological treatments for schizophrenia work by altering the transmission of _____, a neuromodulator that alters neuron-to-neuron communication. ([p. 408](#))
12. Short delays are used in the _____ task for the purpose of assessing working memory; longer delays are used in the same task for the purpose of studying long-term

memory. ([pp. 377–378](#))

13. Place-based models of memory are also known as _____ models of memory because they imply the existence of two or more different places for memories to be stored; similarly, state-based models of memory have been referred to as _____ models of memory because they imply that there is only one place for memory, although these memories can be in various states. ([p. 379](#))
14. The _____ task consists of a series of names of colors, each printed in a color that is *different* from the color being named. ([p. 387](#))
15. General _____, the capacity for learning, reasoning, and understanding, appears to be associated with a strong _____ memory, especially for manipulating larger numbers of rules, concepts, goals, and ideas. ([pp. 388–389](#))
16. In humans, the prefrontal cortex encompasses approximately _____ of the cerebral cortex. ([p. 391](#))
17. Earl Miller argued that the key “cognitive” contribution of the prefrontal cortex to working memory is the ability to sustain activity despite _____. ([p. 397](#))
18. Frontal-lobe patients with damage to the _____ side are most likely to show specialized deficits in verbal (as opposed to visuospatial) working memory. ([p. 400](#))
19. _____ is one of several enzymes that are critical for degrading dopamine. ([p. 408](#))

Concept Check

1. Juan chats with a pretty girl at a party. She tells him her phone number is (617) 555-1812, extension 2001, but he has no way to write it down. How can Juan remember the 14 numbers of her phone number until he can find a pencil and paper?
2. Describe two aspects of executive control that are used both in driving a car and in talking on a cell phone.

3. If you could see an image of someone's frontal lobes while he was rehearsing a list of words, would you see more activity on the left side or the right side? What if he was rehearsing visual images?
4. Tanya is trying to concentrate during a neuroanatomy lecture because she really wants to get into medical school, but she keeps noticing Peter's adorable dimples. Which part of her brain is showing sustained attention to the neuroanatomy images, and which part is being distracted by Peter's dimples?
5. Jerry is trying to remember the name of a woman he met, but all he can recall is that her name is similar to the word for a part of her anatomy. As Jerry struggles to recall her name, is he more likely to be activating his anterior or his posterior left ventrolateral prefrontal cortex?
6. Would a person with ADHD be more likely to take up soccer or duck hunting?

[Answers to Test Your Knowledge](#)

Models of Working Memory

Place: A and D

State: B and C

Varieties of Working Memory and Cognitive Control

1. C
2. D
3. A
4. B

Functional Neuroanatomy of the Prefrontal Cortex

1. Monitoring and manipulating information requires the DLPFC.
2. Verbal rehearsal requires the left VLPFC.
3. Semantic encoding is done by the anterior left VLPFC.
4. Phonological encoding is a specialty of the posterior left VLPFC.
5. Visuospatial rehearsal requires the right VLPFC.

Integrative Topics Module

Chapter 10 Emotional Influences on Learning and Memory



Iudex/Getty Images

Behavioral Processes

[What Is Emotion?](#)

[Assessing Emotion in Nonhuman Animals](#)

[Learning About Emotion-Evoking Stimuli: Focus on Fear](#)

[*Learning and Memory in Everyday Life: “Immunizing” Against Learned Helplessness*](#)

[Memory for Emotional Events](#)

[*Learning and Memory in Everyday Life: Truth or Consequences*](#)

Brain Substrates

[Emotional Learning in the Brain](#)

[Conscious Feelings and the Frontal Lobes](#)

[Emotional Memory in the Brain](#)

[*Learning and Memory in Everyday Life: How Much Stress Is Too Much?*](#)

Clinical Perspectives

[Phobias](#)

[Posttraumatic Stress Disorder](#)

ON THE MORNING OF SEPTEMBER 11, 2001, terrorists hijacked four commercial airplanes and crashed them into the Twin Towers in New York City; the Pentagon in Washington, DC; and a field in Pennsylvania. It was the worst terrorist attack in U.S. history, killing more than 2,000 people. Most Americans who are old enough to remember that morning report that they still recall exactly where and when they heard the news. Even those who lived far from the sites may remember in crisp detail how they heard the news on TV or from a friend. These memories persist, nearly two decades later.



Spencer Platt/Getty Images

Highly emotional events such as the 9/11 terrorist attacks can produce exceptionally vivid and long-lasting memories. Do you remember where and how you heard about this, or another highly emotional event? How detailed is your memory? How sure are you of its accuracy?

If you are too young to remember 9/11 personally, maybe you have a vivid memory for where

and how you heard the news about a more recent national tragedy, such as the 2013 Boston Marathon bombing or the February 14, 2018, shooting at Marjory Stoneman Douglas High School, or even a personal tragedy, such as the death of someone close to you. You may be able to remember exactly how you felt, who was with you at the time, what you said, and even what you had been doing before you heard the news. You may feel that you can recall the event “like it happened yesterday,” even if years have passed.

But now try to remember what you were doing on, say, June 10, 2018. Unless something significant happened to you on that day, the odds are that you have few, if any, detailed memories of it. The reason for this difference is emotion: for most people, June 10, 2018, was just another day; in contrast, momentous events such as the 9/11 attacks can arouse intense emotions that help burn the day’s happenings into our memories in vivid and lasting detail.

Why should emotions influence memory so strongly? For one thing, events that arouse strong emotions often teach important life lessons. A rabbit that runs past a wolf in its lair and narrowly escapes being eaten should remember exactly where the encounter occurred, so it can avoid that place in the future. The emotional response of fear caused by encountering the wolf may help signal that something important is happening—something the rabbit needs to remember particularly well. In modern life, few humans encounter wild wolves, but we do experience situations that cause intense fear, as well as other strong emotions such as joy, anger, shame, lust, and surprise. Memories laid down during times of such heightened emotion are more likely to be remembered for longer, and in greater detail, than memories that are not connected with strong emotions.

This chapter explores the relationship between memory and emotion. Much of the time, this is a positive relationship, with emotion promoting faster learning and more enduring memories. But the darker side is that too much emotion can *interfere* with memory and even cause or contribute to clinical disorders—such as depression, stress disorders, and phobias. One of the chief reasons for studying emotion is to understand these linkages—and to help those who suffer from emotions gone awry.

10.1 Behavioral Processes

Suppose a young man, Sammy, is walking home alone late at night and takes a shortcut through a dark alley. He is aware that he is not in the safest of neighborhoods, and he is feeling a little nervous. Suddenly, he hears a noise behind him—possibly an attacker—and he jumps. His heart skips a beat, his hands grow clammy, and his face pales. Breathing heavily, he turns to look behind him for the source of the threat. All these responses are part of the emotional experience of fear. Some emotional responses—like the reaction to a loud noise in a dark alley—are innate, whereas others—like Sammy’s general anxiety as he walks through what he knows to be a dangerous neighborhood—are learned through experience or social transmission.

What Is Emotion?

We all know an emotion when we feel one, yet emotion, like attention and consciousness, is one of those psychological concepts that are maddeningly difficult to define. In this book, we will define **emotion** as a cluster of three distinct but interrelated phenomena: physiological responses, overt behaviors, and conscious feelings. Physiological responses associated with emotion include changes in heart rate, perspiration levels, respiration, and other body functions. Overt (or observable) behaviors associated with emotion include facial expression, vocal tone, and posture. Conscious feelings associated with emotion are the subjective experiences of sadness, happiness, and so on.

emotion

A cluster of three distinct but interrelated sets of phenomena—physiological responses, overt behaviors, and conscious feelings—produced in response to an affecting situation.

Sammy’s physiological responses to walking in the dark alley include an increase in heart rate, an increase in perspiration, and a diversion of blood away from the capillaries in his face (making him appear pale). His overt behaviors include jumping, widening his eyes, and looking around. The conscious feeling is the fear that accompanies his understanding that he is potentially in danger. This cluster of physiological, motor, and conscious reactions constitutes a **fear response**. Other emotions produce different combinations of physiology, behavior, and conscious feelings. For example, a child being asked to eat broccoli may experience a conscious feeling of disgust; her overt behaviors may include sticking out her tongue and poking at her food, and her physiological responses may include a decrease in heart rate.

fear response

A cluster of physiological changes, overt behaviors, and conscious reactions that accompany the emotion of fear. In the laboratory, the physiological changes and motor behaviors are often taken to imply presence of fear whether or not the accompanying conscious experience of fear can be documented.

Emotion researcher Paul Ekman suggested that a small set of distinct emotions are hardwired in humans from birth. This set includes happiness, sadness, anger, fear, disgust, and surprise (**Figure 10.1**; [Ekman & Friesen, 1984](#); [McNally, Bryant, & Ehlers, 2003](#)). Other researchers would include other emotions, such as interest and shame. Researcher Robert Plutchik has suggested that a small set of “basic” emotions can be modified or blended to form complex emotions; much as an artist can combine the primary colors of yellow and blue to form green, so can we blend anger and disgust to form contempt or blend joy and anticipation to form optimism ([Plutchik, 2001](#)).



Ekman & Matsumoto, Japanese and Caucasian Facial Expressions of Emotions

FIGURE 10.1 Faces illustrating “universal” human emotions Humans of every race and culture seem to experience happiness, surprise, fear, sadness, anger, and disgust and can recognize the expression of these emotions in others.

All humans, from all cultures, feel emotions and can recognize overt behaviors (e.g., facial expressions) that signal these emotions in others. For example, Ekman and his colleague Wallace Friesen showed pictures like those in [Figure 10.1](#) to people of an isolated tribe in New Guinea. Although these New Guineans lived in a culture very different from that of industrialized North America, they had no difficulty recognizing the emotions connoted by the facial expressions of

North American college students—and the New Guineans’ facial expressions were equally well understood by North Americans ([Ekman & Friesen, 1971](#)).

This is not to say that all humans manifest (or interpret) emotional displays the same way ([Gendron, Crivelli, & Barrett, 2018](#)). Different cultures may teach their members different rules about the appropriate ways to display emotions in various social contexts. For example, traditional Japanese culture (which places a premium on respect and orderly behavior) encourages suppression of emotional display to a greater degree than does American culture (which tends to value individualism more than social order). Thus, while watching a film containing unpleasant scenes, both American and Japanese students display similar overt behaviors (e.g., grimacing at the nastiest parts), but if an authority figure, such as an experimenter, is present, the Japanese students tend to mask their negative expressions more than American students do ([Ekman, 1992](#)).

Similarly, in many cultures around the world, the physiological responses associated with emotion are more or less the same in men and women, but women are much more likely to exhibit overt behaviors associated with happiness and sadness, perhaps because of cultural rules dictating that “men don’t cry” ([Eisenberg & Lennon, 1983](#); [Kring & Gordon, 1998](#)). In short, the physiological responses and conscious feelings associated with human emotions seem to be innate and universal, but the overt behaviors associated with those emotions may be modified by cultural learning.

Autonomic Arousal and the Fight-or-Flight Response

Imagine you are walking through the forest, and you come face-to-face with a bear. You probably freeze (an overt behavior). But what physiological responses do you experience? Most likely, your heartbeat speeds up, and your respiration rate increases. Fine hairs on your arms and neck may stand on end, and your hands may sweat. Hormones flood your body, causing you to feel a “rush” of energy and excitement. Other bodily responses may be less noticeable to you: digestion halts, your pupils dilate, and extra blood is directed to the muscles in your legs in case you have to run away.

This collection of bodily responses, called [arousal](#), or (more colorfully) the [fight-or-flight response](#), is the body’s way of preparing you to face a challenge or threat: either by fighting or

by running away. Blood pressure and heart rate increase, and blood flow—with its cargo of glucose that provides energy—is diverted toward the body systems that are most likely to help you in this effort, including your lungs and the muscles in your legs ([Table 10.1](#)). Other systems, such as digestion and the immune system, are temporarily deprived of energy. These latter systems are important for long-term survival—but if you don’t survive the immediate threat, it doesn’t really matter whether you digested that last meal or fought off that cold.

TABLE 10.1 Some Components of the Fight-or-Flight Response

Increases in ...	Decreases in ...
Blood pressure and heart rate	Digestion
Respiration	Immune system function
Blood glucose level	Sexual arousal
Pain suppression	Touch sensitivity
Perception and awareness	Peripheral vision
Blood flow to large muscles in legs and arms	Growth

arousal (fight-or-flight response)

A collection of bodily responses (including increased blood flow to muscles, increased respiration, and depressed digestion and immune function) that prepare the body to face a threat.

The physiological components of arousal are mediated by the [autonomic nervous system \(ANS\)](#), which is a part of the peripheral nervous system (discussed in [Chapter 2](#)). The ANS consists of nerves and structures that control internal organs and glands. The word *autonomic* in “autonomic nervous system” means that the ANS can operate autonomously, without conscious control. When the brain senses a challenge or threat, the ANS signals the adrenal glands to release [stress hormones](#), which act throughout the body to turn the fight-or-flight response on and off. One formal definition of [stress](#) is any event or stimulus that causes bodily arousal and the release of stress hormones. Major stress hormones include [epinephrine](#) (also called [adrenaline](#)) and [glucocorticoids](#). The chief glucocorticoid in humans is [cortisol](#). In the short term, our reactions to stress through arousal and release of stress hormones prepare us to deal with the challenge before us; usually, once the challenge is past, the ANS turns off the fight-or-flight response, and stress hormone levels drop back to normal.

autonomic nervous system (ANS)

A collection of nerves and structures that control internal organs and glands.

stress hormone

A hormone that is released in response to signals from the autonomic nervous system (ANS) and helps mediate the fight-or-flight response; examples include norepinephrine and the glucocorticoids.

stress

Any stimulus or event that causes bodily arousal and release of stress hormones.

epinephrine (adrenaline)

A stress hormone that helps mediate the fight-or-flight response.

glucocorticoid

Any of a class of stress hormones (including cortisol in humans) that help to mediate the fight-or-flight response.

cortisol

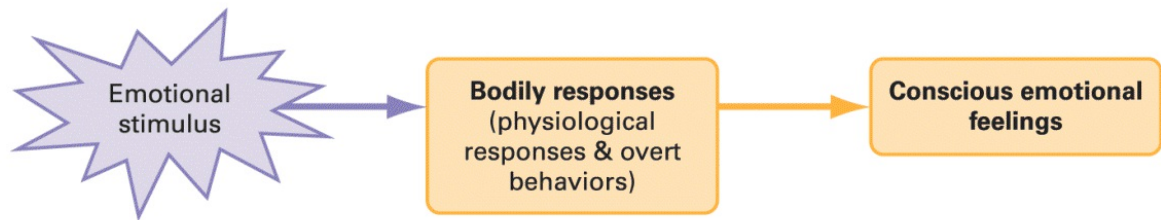
The chief glucocorticoid in humans.

Strong pleasant emotions, such as happiness and surprise, can cause physiological arousal that is very similar to the components of the fight-or-flight response. Thus, a young man in the throes of sexual arousal may experience many of the same physiological responses (dilated pupils, increased heart rate and blood pressure, sweating, and so on) as one who is suddenly attacked by a masked gunman. Many of the same features of arousal occur in a range of emotions, although there are differences. For example, heart rate increases during many emotions but falls during disgust ([Levenson, 1992](#)). So why do we experience increased heart rate and blood pressure as components of a pleasant emotion in one situation and as part of a negative emotion in another? Several theories have been proposed.

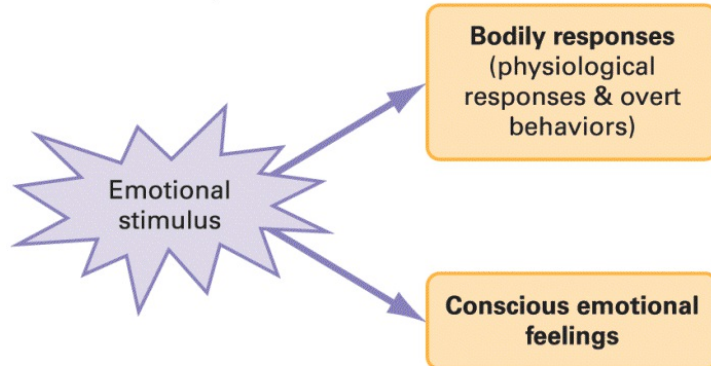
James–Lange Theory of Emotion

In the late nineteenth century, William James (see [Chapter 1](#)) proposed a theory of emotion that says conscious feelings of emotion occur when the mind senses the physiological responses associated with fear or some other kind of arousal. A very similar idea was proposed at about the same time by the Danish physiologist Carl Lange, so the theory is now usually called the **[James–Lange theory of emotion](#)** ([LaBar & LeDoux, 2003](#)). **Figure 10.2A** illustrates this theory: certain stimuli evoke bodily responses in the form of physiological changes and overt behaviors; our mind then interprets these bodily responses, producing conscious feelings of emotion. Several modern researchers have developed related theories, often called **[somatic theories of emotion](#)**, all based on this central premise that bodily responses to stimuli come first, and these determine or induce emotions ([Damasio, 1996](#); [Zajonc, 1980, 1984](#)).

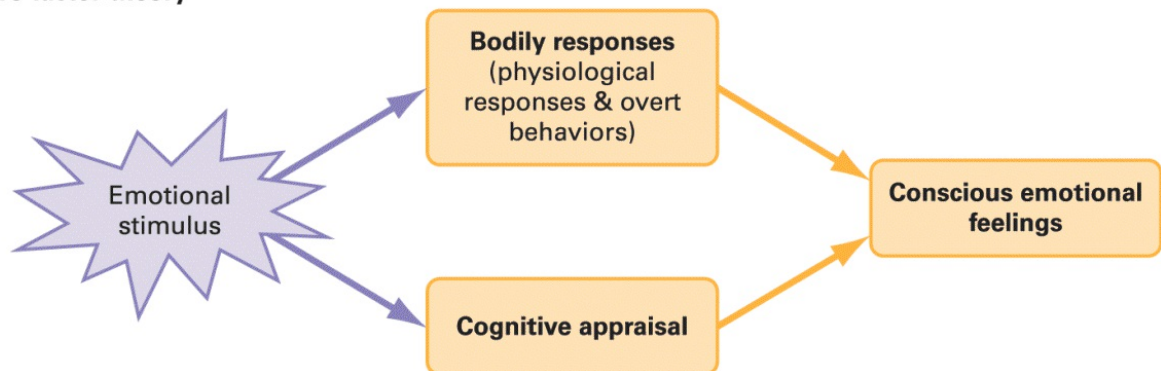
A James–Lange theory



B Cannon–Bard theory



C Two-factor theory



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

FIGURE 10.2 Theories of emotion (A) The James–Lange theory and other somatic theories of emotion posit that, when we encounter an emotion-arousing stimulus, bodily responses (including physiological responses and overt behaviors) come first, and these then trigger our conscious feelings of emotion. (B) The Cannon–Bard theory posits that bodily responses and conscious emotional feelings occur simultaneously and independently. (C) The two-factor theory of emotion posits that conscious feelings of emotion are produced through interaction between our bodily responses and our cognitive appraisal of the situation.

Description

James-Lange theory: Emotional stimulus leads to bodily responses (physiological responses and overt behaviors), which lead to conscious emotional feelings.

Cannon-Bard theory: Emotional stimulus leads to bodily responses (physiological responses and overt behaviors) and conscious emotional feelings.

Two-factor theory: Emotional stimulus leads to bodily responses (physiological responses and overt behaviors) and cognitive appraisal. These two lead to conscious emotional feelings.

James–Lange theory of emotion

The theory that conscious feelings of emotion occur when the mind senses the physiological responses associated with fear or some other kind of arousal.

somatic theories of emotion

Theories of emotion based on the central premise that physiological responses to stimuli come first, and these determine or induce emotions.

One prediction of somatic theories of emotion is that researchers should be able to evoke a given emotion in a person just by inducing the corresponding bodily responses. In fact, there is some evidence that this is so. In one series of experiments, researchers were able to elicit moods in volunteers just by asking the volunteers to move various facial muscles (without telling them what mood this was intended to produce). For example, one instruction went as follows: “Pull your eyebrows down and together; raise your upper eyelids and tighten your lower eyelids; narrow your lips and press them together.” How do you feel when you make this expression and hold it for a few seconds? In case you couldn’t guess, these facial contortions were meant to simulate anger. Volunteers who made such a face generally reported feelings of mild anger or annoyance ([Ekman, 1992](#)).

On the other hand, when volunteers were told to turn up the corners of their mouths and crinkle their eyes, they reported a mild overall increase in feelings of happiness ([Ekman, 1992](#)). Many studies have reported that participants engaging in such “smile-inducing” manipulations often judge cartoons to be funnier ([Dzokoto, Wallace, Peters, & Bentsi-Enchill, 2014](#); [Soussignan, 2002](#); [Strack, Martin, & Stepper, 1988](#)) and exhibit faster physiological recovery from stress ([Kraft & Pressman, 2012](#)). These results have been challenged by a recent large-scale replication attempt by 17 research teams around the globe, in which only about half of the teams reported any effect of facial manipulations on how funny their participants found a set of cartoons ([Acosta et al., 2016](#)). Still, “mood-induction” studies provide some support for somatic theories of emotion, which posit that our conscious feelings of emotion depend on what our body is telling us: bodily responses come first, conscious feelings second.

Cannon–Bard Theory of Emotion

Walter Cannon, a former student of William James, thought that his mentor had it backward. People, Cannon argued, can experience physiological arousal without necessarily experiencing conscious feelings of emotion. For example, after running, a person may experience a racing heart, but this does not mean she will feel afraid. In addition, as you read earlier, many emotion-provoking stimuli give rise to similar sets of physiological responses: fight (anger), flight (fear), and sexual excitement can all cause increases in heart rate, perspiration, and hormone release. How, then, does your conscious mind determine which emotion is being signaled by the increased heart rate and other physiological responses?

Cannon's proposal, which was later extended by Philip Bard and became known as the **Cannon–Bard theory of emotion**, was that bodily responses and conscious emotional feelings occur simultaneously and independently, with neither causing the other ([Cannon, 1927](#)). [Figure 10.2B](#) illustrates this idea. Emotion researcher Joseph LeDoux recently proposed a similar view, arguing that the brain systems mediating the physiological and behavioral components of emotion are separate from the brain systems that mediate conscious feelings ([LeDoux & Pine, 2016](#)).

Cannon–Bard theory of emotion

The theory that conscious feelings of emotion and bodily responses (physiological responses and overt behaviors) occur simultaneously and independently.

Two-Factor Theory of Emotion

A few decades after Cannon's proposal, Stanley Schachter and Jerome Singer came up with a theory of emotion that incorporated aspects of both the James–Lange and Cannon–Bard theories. Schachter and Singer said the James–Lange theory was partly correct, in that arousal is an important determinant of emotion; but they also said the Cannon–Bard theory was partly correct too, in that arousal alone isn't sufficient to determine what we feel.

In a famous experiment, Schachter and Singer injected volunteers with epinephrine, one of the stress hormones that help produce physiological arousal (increased heart rate, increased blood pressure, and so on). They then put each volunteer in a room with a person who (unbeknownst to the volunteer) had been instructed to act in a certain way. The volunteers began to catch the mood of these accomplices: when paired with accomplices who were acting joyful, the

volunteers began to act happy, too; when paired with accomplices who were acting angry, other volunteers began to act annoyed themselves. In other words, epinephrine injections caused physiological arousal, which each volunteer's brain interpreted according to the context in which that individual had been placed ([Schachter & Singer, 1962](#)). It appears that when the same ambiguous bodily responses can occur in different emotions, the label we apply to the responses (e.g., “joy” or “anger”) depends on the situation we are in.

These results led Schachter and Singer to propose a **[two-factor theory of emotion](#)**, which posits that a combination of cognitive appraisal and perception of bodily responses together determine our conscious emotional feelings ([Figure 10.2C](#)). In other words, if a young man is alone in the woods and sees a bear, cognitive appraisal warns him of the danger, and this appraisal interacts with his physiological arousal to produce the conscious emotional feeling of fear. In contrast, the same young man meeting his girlfriend after a long absence may have a similar pattern of physiological arousal (rapid heartbeat, dilated pupils), but his cognitive appraisal of the situation will lead to very different conscious emotional feelings: happiness and excitement. On the one hand, as James proposed, the particular pattern of bodily responses contributes to our conscious feelings of emotion; on the other hand, Schachter and Singer argued, our cognitive awareness helps us interpret these bodily responses in accordance with our current context.

two-factor theory of emotion

The theory that a combination of cognitive appraisal and perception of biological changes together determines our conscious feelings of emotion.

Another famous experiment supporting the two-factor theory of emotion was the “High Bridge” study ([Dutton & Aron, 1974](#)), in which a female researcher, stationed on a footbridge, asked male passersby to complete a brief survey. In some cases, the footbridge was a low, safe-looking structure; in others, it was a swaying cable bridge that spanned a deep ravine (the Capilano Suspension Bridge in Vancouver). After each participant completed the survey, the researcher would give him her phone number “in case you have further questions.” The researchers predicted that men on the dangerous-looking bridge would be highly aroused, likely to misinterpret this feeling as sexual arousal, and therefore more likely to call the woman back looking for a date, compared with men who took the survey while on the safe-looking bridge.

The results? A few of the men who had been tested on the low bridge called the experimenter back, but fully 50% of the men tested on the high bridge phoned. In contrast, when the study was

repeated with a male survey taker, there were hardly any follow-up phone calls from males tested on either bridge, allowing the researchers to rule out the possibility that participants tested on the high bridge were simply more interested in the scientific details of the experiment.

The two-factor theory may also help explain the popularity of horror movies, at which viewers expect to be made to scream in terror. Given that fear is a “negative” emotion, why would otherwise rational people stand in line and pay good money to experience it? Part of the answer is that the strong physiological responses caused by a terrifying movie are not so different from the strong physiological responses caused by intense joy or sexual pleasure. Viewers know they are relatively safe in the context of the movie theater, and this cognitive assessment allows them to interpret the strong arousal as pleasurable rather than threatening.



Left: Ryan McVay/The Image Bank/Getty Images; right: SUZANNE PLUNKETT/AP Images

The moviegoers on the left appear to be enjoying the experience of physiological responses and overt behaviors normally associated with fear and excitement; in contrast, the New Yorkers on the right, fleeing the scene of the 9/11 World Trade Center attacks, do not appear to be enjoying the fear and excitement. How would the two-factor theory of emotion explain the difference in emotion seen in these two photos?

Description

The first photo shows a group of people wearing 3-D glasses and screaming with excitement. The second photo shows New Yorkers fleeing the site the attack on the World Trade Center on September 11, 2001.

TEST YOUR KNOWLEDGE

Theories of Emotion

Janet is at a music concert featuring her favorite band. As the lights dim and the band walks onstage to a crashing opening

chord, she jumps to her feet and cheers along with the rest of the crowd, her heart racing with excitement. How would each of the following theories of emotion describe this situation? ([Answers appear at the end of the chapter.](#))

1. James–Lange theory
2. Cannon–Bard theory
3. Two-factor theory

Assessing Emotion in Nonhuman Animals

So far, we have discussed emotions as defined in and by humans. Can other animals experience emotion? Many researchers believe that they can.

The literature on animal behavior is rich with examples that seem to describe animal emotions. Poachers shoot a female elephant; the other elephants in her tribe try to revive her by propping her up and sticking grass in her mouth. When she dies despite their efforts, they sprinkle earth and branches over the body, and her daughter returns to the site periodically thereafter and strokes the bones with her trunk ([Moss, 1988](#)). A female chimpanzee gives birth; as the baby is delivered, the chimpanzee who is the mother's best friend shrieks and embraces nearby chimpanzees, then spends the next few days caring for mother and baby ([de Waal, 1996](#)). A killer whale grows sick; his podmates flank him and protect him, helping him stay near the surface, where he can breathe, even guiding him into shallow waters at the risk of beaching themselves, refusing to abandon him until he finally dies ([Porter, 1977](#)).

Can elephants feel sadness, monkeys feel empathy, and killer whales feel sympathy? Or are the animals described here simply acting on blind instinct, without any of the emotional overtones we would attribute to humans? The fact is that we may never know if other animals experience subjective emotional feelings, or if they do, what those feelings are like. But remember that subjective feelings are only one component of emotions. The other two components, physiological responses and overt behaviors, are certainly apparent and can be studied in nonhuman animals.

Fear Responses Across Species

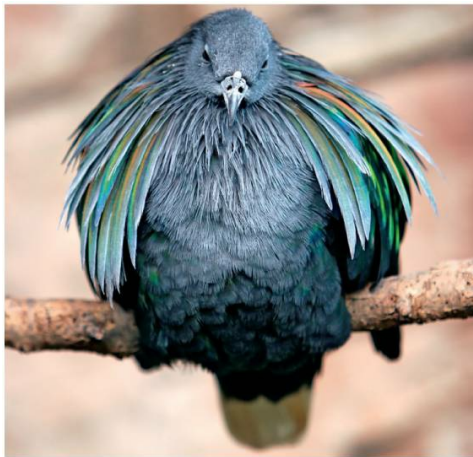
As early as the mid-nineteenth century, Charles Darwin noted that many species of animals react

to arousing stimuli in similar ways ([Darwin, 1872/1965](#)). For example, when a gorilla encounters a frightening stimulus, its first reactions may include a sudden jump (“startle”) followed by a period of alert immobility (“freezing”). It may display **piloerection**, meaning that its body hair stands on end; this makes the animal look bigger and more threatening. In extreme situations, the gorilla may defecate or urinate. These responses are remarkably similar to Sammy’s physiological reactions in the dark alley: hearing a suspicious noise makes him jump and then freeze while he assesses the situation. Tiny hairs on the back of Sammy’s neck and arms may stand on end, and he may develop goose bumps, which Darwin suggested are the remnants of our hairy ancestors’ piloerection response. In extreme circumstances, Sammy may even be terrified enough to lose bladder or sphincter control. All of these fear reactions in humans seem to have direct analogues in other primates.

piloerection

A fear response in mammals in which body hair stands on end, making the animal look bigger and more threatening than it is.

Similarly, a startled rat shows changes in blood pressure, heart rate, and hormonal release that are much like the changes seen in cats, rabbits, monkeys, and humans. Even fish, reptiles, and birds show some of these responses. For example, many birds fluff their feathers when presented with a threatening stimulus, and some fish spread their fins wide or puff up their bodies (like the pufferfish in the photo at the beginning of this chapter), which makes them look larger, in a defensive adaptation analogous to piloerection in mammals. When such reactions appear, researchers report that the animal is expressing physiological and behavioral components of a fear response, even if we can’t be sure whether the animal is consciously feeling fear in the same way that a human might.



Left: Nick Stubbs/Shutterstock; right: Eliot Lyons/Nature Picture Library

Emotional displays in animals often have striking similarities to displays of human emotion. For example, when startled, many animals—including fish, birds, and mammals—will puff themselves up, fluff their feathers, or bristle their fur in an apparent attempt to make themselves look bigger (and scare off predators). What emotional displays in humans share some features with emotional displays seen in animals?

It is important to keep in mind that physiological responses and overt behaviors do not automatically equate with emotions in humans, either. Remember the woman whose heart races after running but who does not feel fear? Another example comes from research in which human participants are trained to make a response to predict or avoid an unpleasant event such as electrical stimulation (shock). Here, ethical concerns typically dictate that participants be allowed to set the intensity of the shock at a level they experience as “unpleasant but not painful.” Under these conditions, humans may experience physiological arousal, but they typically do not report conscious feelings of fear while anticipating upcoming shock ([LaBar, 2009](#)). So when studying human participants, just as with rats and other animals, researchers evoking and studying emotional responses need to remember that physiological responses and overt behaviors are consistent with—but not necessarily proof of—conscious feelings of emotion.

Fight-or-Flight or Tend-and-Befriend?

Just as animals of different species may respond to threatening stimuli with different defensive behaviors, there may also be differences among individuals of the same species. Specifically, Shelley Taylor and her colleagues have argued that, rather than fight-or-flight, some individuals are more likely to respond to threat with a strategy nicknamed **tend-and-befriend** ([Taylor, 2000](#)): protecting offspring (tending) and seeking out social support (befriending).

tend-and-befriend response

A response to threat that involves tending to offspring (tending) and seeking out social support (befriending).

For example, a rat pup removed from the cage and handled by humans will exhibit a physiological stress response; upon its return, the mother will spend time licking and grooming (tending) the pup, a behavior that reduces physiological stress responses in both the mother and pup. Similarly, among humans and nonhuman primates, holding and nurturing an infant during times of stress can produce reductions in physiological stress responses in both the mother and her child.

Befriending may also be a form of social interaction that produces relief from stress. For example, when a female chimpanzee is the victim of aggression, her female friends will often spend time sitting near her and grooming her (befriending), a behavior that again reduces physiological stress in both animals.

Taylor and colleagues argued that females are particularly likely to show tend-and-befriend responses. Thus, among female rats, levels of stress hormones drop when they are placed in a cage with other females; on the other hand, male rats typically show higher levels of stress hormones, and more aggression, when placed in a cage with other males. And among humans, women across many cultures are much more likely than men to report providing support to other women (and men) in times of stress, such as job-related difficulties or severe illness ([Tamres, Janicki, & Helgeson, 2002](#)).

This is emphatically not to suggest that males are incapable of caring for their offspring or seeking out social support, nor that females are incapable of displaying aggression; men can and do care for their offspring, and women can and do engage in aggressive behavior. Still, proponents of these ideas suggest that whereas “fight-or-flight” may be a prototypical or default response to threat for many male mammals, “tend-and-befriend” may be a more prototypical female response to threat.

Why might “tend-and-befriend” be an effective response for a female in a threatening or stressful situation? An evolutionary explanation is that, in species where the mother invests considerable time and energy in rearing her offspring, females who protect and nurture their offspring in times of stress may increase the chance that those offspring survive, to pass on their genes in turn. Conversely, mothers who face threat by fighting (risking death or injury) or fleeing (abandoning their offspring) may decrease the offspring’s chances of survival. In addition, females who befriend or affiliate with other females in distress may increase the chances that those females will reciprocate later—a form of reciprocal altruism, as discussed in [Chapter 5](#)—and this may increase the survival chances of both mother and offspring.

There is some biological basis for these ideas. Just as stress hormones such as epinephrine promote components of the fight-or-flight response (as shown in [Table 10.1](#)), so the hormone *oxytocin* promotes behaviors such as maternal tending and social bonding ([Donaldson & Young,](#)

[2008](#); [Insel, 1997](#)). Among its other effects, oxytocin has an anxiolytic effect, meaning that it reduces fear and anxiety and promotes social interaction. In rats, infusions of oxytocin stimulate maternal behavior, even in virgin females who would normally ignore or attack pups; and in humans (both male and female), intranasal spray containing oxytocin can increase interpersonal trust and forgiveness following social rejection (for review, see [Donaldson & Young, 2008](#)). Although oxytocin is present in both male and female mammals, it is enhanced by estrogen (the principal sex hormone in females) but inhibited by testosterone (the principal sex hormone in males). The net effect may be that females are more likely to respond to threat with caregiving and social affiliation than with fighting or withdrawing.

The concept of tend-and-befriend remains controversial; critics argue that the idea of men and women having different biological responses to stress reflects sexual stereotyping (e.g., [Geary & Flinn, 2002](#)). But a general conclusion from all the findings to date is that any individual's response to a specific threat reflects a complex interplay of factors, including hormones and situational variables, that can produce wide variations in patterns of emotional responding, between individuals and even in the same individual at different points in time.

Beyond Fear

Because responses to threatening stimuli are relatively easy to induce, detect, and record, the vast majority of studies on emotion and its effects on learning and memory have focused on how animals learn about and respond to threatening stimuli. But in recent years, increasing attention is being paid to the other emotions as well. [Chapter 5](#) describes how both human and nonhuman animals give strikingly similar “yum” and “ugh” responses to pleasant and unpleasant tastes ([Berridge & Robinson, 1998](#)). In particular, rats that taste sweetness show rhythmic movements of the mouth and protrusion of the tongue—just like humans tasting something pleasant (“yum!”); rats given a bitter taste show mouth gapes, head shakes, and wiping of the face with the paws—just like humans shuddering at a disgusting taste (“ugh!”). Again, we cannot be sure that the rat is experiencing conscious feelings of pleasure or disgust, but these physiological responses and overt behaviors seem remarkably consistent across mammalian species.

Another way in which humans express pleasure, particularly within a social context, is through the overt behavior of laughter. There is now accumulating evidence of laughter-like responses in other mammals, too. For instance, tickling is rewarding to young rats, in the sense that they will

perform behaviors such as running mazes and pressing levers in order to obtain tickle reinforcement ([Burgdorf & Panksepp, 2001](#)). During tickle, rats tend to emit ultrasonic vocalizations that share many acoustic features with the sounds of human laughter ([Panksepp & Burgdorf, 2003](#)). Tickle-induced vocalizations can also be elicited in chimpanzees and gorillas ([Davila-Ross, Owren, & Zimmerman, 2009](#)). Even dogs emit a characteristic “laugh” vocalization during play, and one study has suggested that dogs in a kennel experience reduced stress if exposed to recordings of dog-laugh vocalizations ([Simonet, Versteeg, & Storie, 2005](#)).

The presence of laughter-like vocalizations in rats, dogs, and apes appears to be a social expression of joy. Thus, young rats emit laughter-like vocalizations during play behavior, and they prefer to spend time with older animals that “laugh” frequently ([Panksepp & Burgdorf, 2003](#)). Although the study of nonhuman laughter is still relatively young, there is already some evidence that the same brain regions that are active during social laughter in humans are also active in these other animals during laughter-like responses ([Meyer, Baumann, Wildgruber, & Alter, 2007](#)). Thus, recording laughter-like vocalizations may be one way researchers can measure joy responses in nonhuman animals. Again, we cannot be sure that “laughing” animals are experiencing conscious feelings of joy, only that their physiological responses and overt behaviors are similar to those exhibited by happy humans as they laugh in delight.

Learning About Emotion-Evoking Stimuli: Focus on Fear

Further research into joy responses in animals may produce insights into how nonhuman animals learn joy responses and whether it differs from how they learn fear responses. In the meantime, however, as stated previously, the vast majority of studies on emotional learning in nonhuman animals have focused on how animals respond to threatening stimuli. This is not because fear is inherently more important or interesting than the other emotions but simply because researchers have long felt confident about their ability to elicit and recognize fear responses in nonhuman animals. The learning of these emotional responses, including both overt behavior and physiological changes, can be particularly fast, strong, and long-lasting.

Conditioned Emotional Responses: Learning to Predict Danger

When a rat is given an unpleasant surprising stimulus, such as an unexpected electric shock, it typically displays a short period of alert immobility: the freezing response that was described earlier as also occurring in humans and gorillas. Responding to a potential threat by staying very still may help a small animal avoid predators, which often have visual systems tuned to detect movement. It may also allow an animal of any size to allocate full attention to specific sensory inputs to help determine what and where the threat is. After a moment, if nothing further happens, the animal typically relaxes and goes back to what it was doing before.

The freezing response is not learned; rather, it is an innate fear response to a threatening situation. In this example, the freezing behavior is an unconditioned response (UR) evoked by the shock, which is an unconditioned stimulus (US). ([Chapter 4](#) introduces classical conditioning and the concepts of CS, US, CR, and UR.) In addition to freezing behavior, the shock US also evokes autonomic URs, such as increases in blood pressure and heart rate. However, if the shock US is repeatedly preceded by a neutral conditioned stimulus (CS)—say, a tone or a light—the animal will learn the CS–US association and may then produce conditioned responses (CRs) to the CS.

Such CRs are called **conditioned emotional responses**. [Figure 10.3](#) shows an example of conditioned emotional responding in rats. In this example, researchers first play a 10-second tone CS. The rats respond with a brief freezing response as they listen to the unfamiliar sound ([Figure 10.3A](#)). Next, the tone is paired with a foot-shock US for one or more trials. Later, the tone is presented alone; now, the rat expects the foot shock to follow, and the rat manifests this learning through a behavioral CR: prolonged freezing to the tone in anticipation of the upcoming shock ([Figure 10.3A](#)).

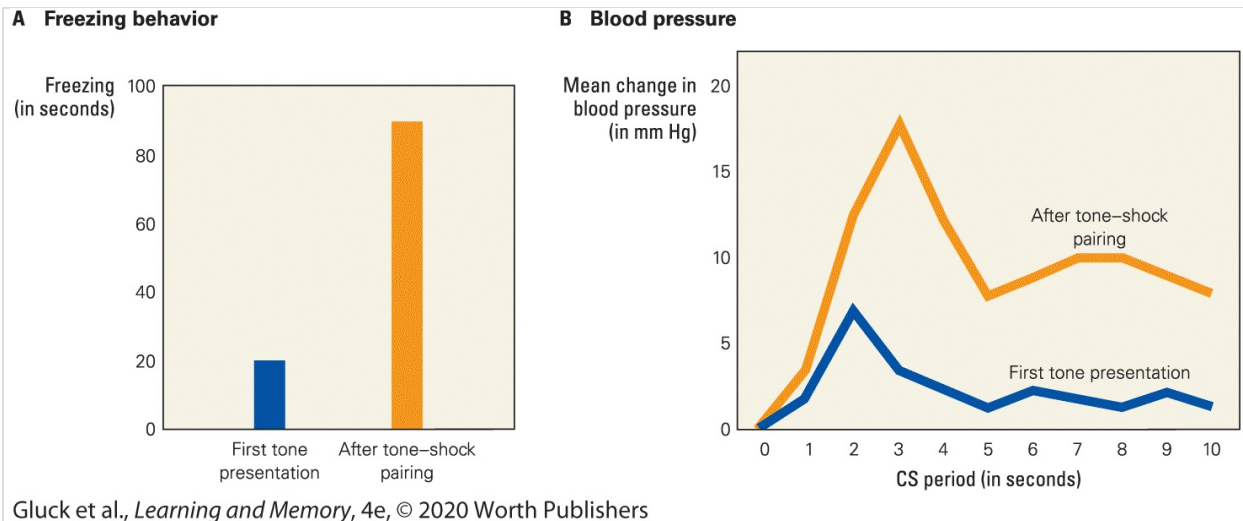


FIGURE 10.3 Two types of conditioned emotional responding in rats (A) On first presentation, a 10-second tone may trigger a brief freezing response; but, after the tone has been paired with a foot-shock US, it evokes a strong freezing response (a fear CR). (B) Similarly, the first tone presentation evokes only a mild and transient increase in blood pressure, but after tone–shock pairing, the tone CS evokes a strong and persisting increase in blood pressure. Blood pressure change and freezing are conditioned emotional responses, indicating that the rat has learned the CS–US association.

Research from [LeDoux, 1993](#).

Description

The bar graph shows the freezing time of first tone presentation and after tone-shock presentation in seconds. The readings on the vertical axis range from 0 to 100 in increments of 20. The data in the graph reads as follows. first tone presentation-20 seconds, after tone-shock pairing-95 seconds.

The second graph plots mean change in blood pressure in millimeters of Hg on the vertical axis against C S period in seconds on the horizontal axis. The readings on the vertical axis range from 0 to 20 in increments of 5 and the readings on the horizontal axis range from 0 to 10 in increments of 1. The graph has two curves representing first tone presentation and after tone-stock pairing. The first tone presentation curve starts from (0,0), increases steeply to a peak at (2,7) after which it descends gradually, ending at (10,2). The after tone-shock pairing curve starts from (0,0), increases steeply to a point at (3, 17.5), after which it descends steeply to a point at (5,7). After the point at (5,7), it ascends gradually to (7,10), plateaus for a while and descends after that.

conditioned emotional response.

A classically conditioned response (CR), such as physiological arousal, that is produced in response to a CS that has been paired with an emotion-evoking US.

In addition to the freezing CR, which is an overt behavior, physiological changes can become part of the conditioned emotional response to the stimulus associated with threat. Thus, the first time the tone is presented, it evokes a mild, temporary increase in blood pressure ([Figure 10.3B](#)); after tone–shock pairing, the tone evokes a sharp rise in blood pressure that persists through the

duration of the tone. If the experimenters had chosen to measure other bodily responses indicative of a fight-or-flight response—such as increased heart rate, stress hormone release, or defecation—they would probably have found that the tone elicited many of these responses as well, as further components of the conditioned emotional response. These learned fear responses are similar to the behaviors that rats exhibit when confronted with a natural threat stimulus, such as the scent of a cat ([Fendt & Fanselow, 1999](#)).

The same basic rules of conditioned emotional responding apply in other species, too. For example, [Chapter 4](#) discusses the sea snail *Aplysia*, which can learn to withdraw its gill (CR) in response to a gentle touch (CS) that predicts an upcoming tail shock (US). Is the sea snail “feeling” fear in the same way as a rat huddling in a corner of its cage or as a New Yorker trying to flee the scene of the terrorist attacks on 9/11? We cannot be sure. All we can know is that the overt behavior of *Aplysia* is a conditioned emotional response not so different from the overt behavior shown by a caged rat displaying freezing behavior or by a fleeing human.

Conditioned freezing in rats may occur after only a single CS–US pairing, and even lowly *Aplysia* can learn a conditioned gill withdrawal in about four trials. Compare that with the classical conditioning of eyeblink responses, as described in [Chapter 4](#)—which may require several hundred trials in rats and rabbits and dozens of trials in humans—and the inescapable conclusion is that emotional learning occurs *quickly*.

Another characteristic of conditioned emotional responses is that they can be very long-lasting and hard to extinguish. It may take many *extinction* trials, in which the animal receives presentations of the CS alone with no US, before the animal stops giving a conditioned emotional response. Even then, extinction does not eliminate the learned response; it only reduces the likelihood that the CS will elicit the CR. For this reason, conditioned emotional responses are very easily reinstated after extinction: sometimes, merely placing the animal back in the experimental chamber where it experienced the US is enough to restore conditioned emotional responding ([Bouton & Peck, 1989](#)).

Conditioned Escape: Learning to Get Away from Danger

Learning to recognize which stimuli signal upcoming danger is a good thing, but getting away

from the danger is even better. In [conditioned escape](#), animals learn to make particular responses in order to escape from or terminate an aversive stimulus. For instance, a rat in an operant conditioning chamber might learn to press a lever in order to terminate a series of mild foot shocks, or a rat placed in a pool of water might learn to locate and climb onto a submerged platform in order to escape from the water. (Rats are quite competent swimmers, but just like humans at the beach, if the water is chilly, they prefer to get out of it.)

conditioned escape

An experimental design in which animals learn to make particular responses in order to escape from or terminate an aversive stimulus.

Escape learning is a form of operant conditioning. [Chapter 5](#) describes operant conditioning as an association in which a discriminative stimulus S^D evokes a behavioral response R, leading to an outcome O:

Discriminative stimulus S^D → Response R → Outcome O

In the shock example, the discriminative stimulus S^D is the initiation of shock, the response R is lever pressing, and the outcome O is escape from shock. This is an example of *negative reinforcement*: the response causes something aversive (shock) to be subtracted from the environment, so the frequency of the response increases. Similarly, in the pool example, S^D is the water, R is locating and climbing onto the platform, and O is escape from the water; this is another example of negative reinforcement in which the response causes something aversive (chill) to be subtracted from the environment, so the frequency of the response increases.

Conditioned escape learning can occur very quickly; the first time the rat is placed in the pool, it may swim around randomly until it locates the submerged platform, but after a few learning experiences, a rat placed back in the pool will typically swim straight to the platform and climb out of the water.

Conditioned Avoidance: Learning to Avoid Danger Altogether

Even better than escaping from danger is learning to avoid it altogether. In [conditioned avoidance](#), animals learn to make particular responses to avoid or prevent arrival of an aversive

stimulus. For example, a rat may be placed in a chamber with a response lever. Periodically, a series of foot shocks is delivered, and if the rat presses the lever when this is happening, it can terminate (escape from) the shocks. In addition, a warning signal, such as a tone, is played just before the foot shocks start; if the rat presses the lever before the tone is finished, the foot shocks are prevented (avoided) altogether. Typically, a rat first learns how to escape from the shocks, then later learns how to avoid them by pressing the lever while the warning signal plays ([Beck et al., 2011](#)).

conditioned avoidance

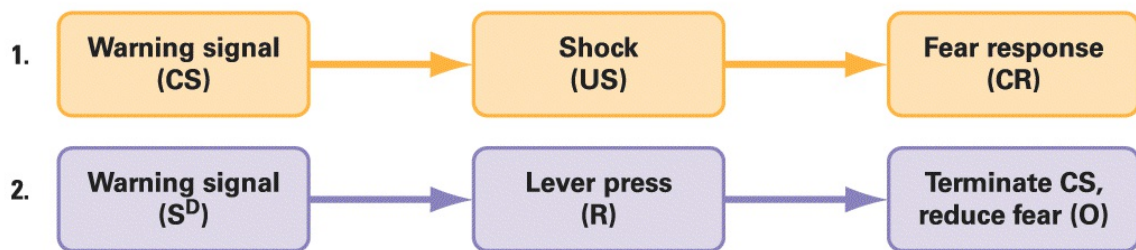
An experimental design in which animals learn to make particular responses to avoid or prevent exposure to an aversive stimulus.

At first glance, it may appear that avoidance learning is simple operant conditioning: the warning signal (S^D) triggers the response (R), which causes avoidance of the shocks (O). Since shocks are aversive, avoidance of the shocks reinforces the response, making the organism more likely to repeat the response in future.

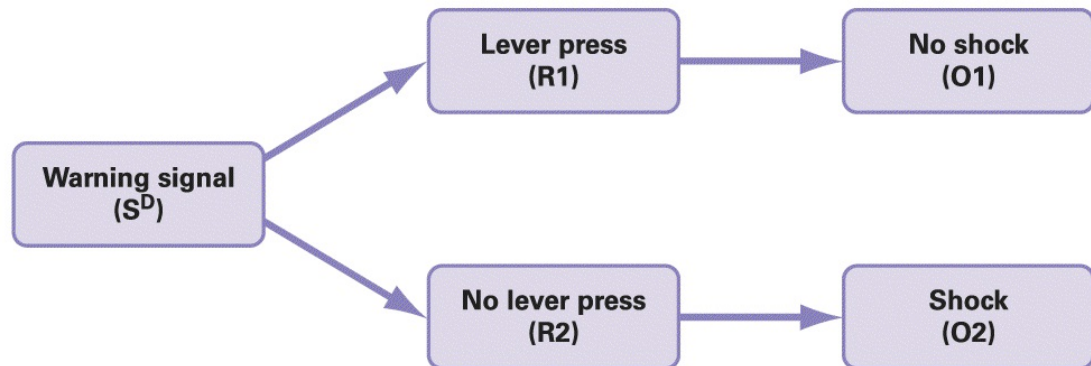
But this simple explanation doesn't quite hold up. Once the animal learns to make the avoidance response regularly, the shocks no longer occur. Therefore, there is no explicit reinforcement for pressing the lever: since the shocks never arrive, they cannot be subtracted from the environment. And by the principles of operant conditioning, if the response is no longer explicitly reinforced, the response should extinguish. Yet it does not! Rather, avoidance behaviors can be incredibly persistent: animals may keep making the avoidance response long after the US is no longer delivered. This is a paradox that requires further explanation.

Some researchers have proposed a [two-factor theory of avoidance learning](#), which states that avoidance learning involves an interaction between classical and operant conditioning ([Dinsmoor, 1954](#); [Mowrer, 1960](#)). According to this view, the first stage of avoidance learning occurs through classical conditioning: as the tone is paired with the shock, it comes to function as a warning signal and becomes a conditioned stimulus (CS) that evokes a conditioned emotional response (CR) in anticipation of the upcoming shock (US). In the second stage, operant conditioning occurs: the avoidance response (lever pressing) is reinforced because it causes cessation of the warning signal and therefore a reduction in fear ([Figure 10.4A](#)). In effect, animals learn to lever press not in order to avoid the shock but, rather, in order to escape from the warning signal (or, more specifically, from the fear response it produces).

A Two-factor theory



B Cognitive expectancy theory



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FIGURE 10.4 Theories of avoidance learning (A) In the two-factor theory of avoidance learning, (1) rats first learn a classically conditioned fear response to a warning signal (CS) paired with shock (US); (2) then rats learn an operant response to terminate the warning signal and the fear it evokes. (B) In the cognitive expectancy theory of avoidance learning, rats learn that different responses predict different outcomes and use this cognitive expectancy to guide their decision of which response to make.

Description

A. Two-factor theory: 1. Warning signal (C S) leads to shock, which leads to fear response (C R).

2. Warning signal (S superscript D) leads to lever press (R) which leads to terminate C S, reduce fear (O).

B. Cognitive expectancy theory: Warning signal (S superscript D) leads to lever press (R 1) and no lever press (R 2).

Lever press R 1 leads to no shock (O 1) and No lever press (R 2) leads to shock (O 2).

two-factor theory of avoidance learning

A theory that avoidance involves an interaction between classical conditioning and operant conditioning.

There are several problems with two-factor theories of avoidance learning, however ([Dymond & Roche, 2009](#)). One major difficulty is that these theories assume avoidance learning is based on learning to fear the warning signal. But, in fact, as the avoidance response is learned, the

organism's fear response (e.g., physiological arousal) to the warning signal *decreases*. If avoidance depends on a fear response to the warning signal, then as the fear response decreases, the avoidance response should decrease, too. Yet this is not what happens. As noted earlier, even in extinction (when the shock no longer occurs), animals often persist in making the avoidance response.

Various researchers have proposed modifications to the basic two-factor theory to address this and other limitations ([Williams, 2001](#)). For example, cognitive theorists have proposed that animals form **cognitive expectancies** and decide between competing possible behaviors based on those expectancies. According to this view, animals learn the expected outcomes of responding and of not responding and then make a decision based on a comparison between the two expected outcomes ([Figure 10.4B](#); [Dickinson & Balleine, 2000](#); [Seligman & Johnston, 1973](#)).

cognitive expectancy

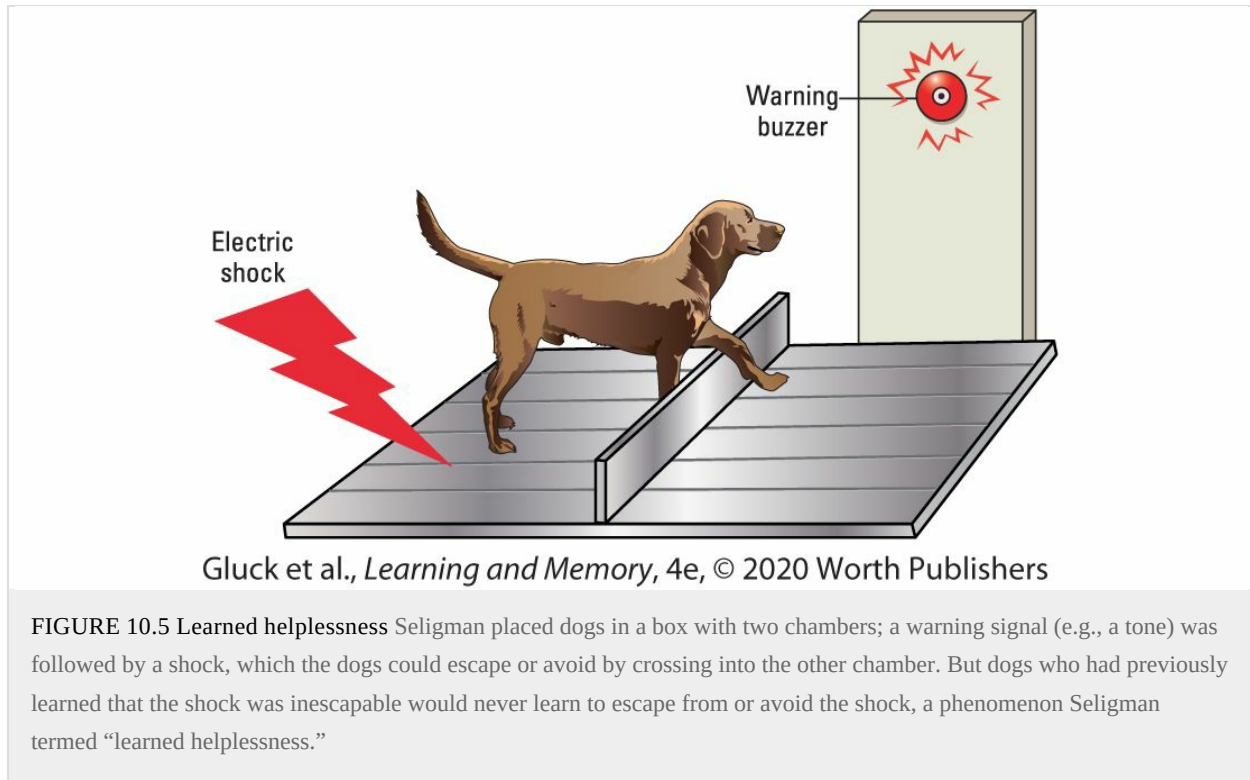
The expected outcome of a response, used as a guide in determining whether or not to make that response.

Some have argued for integrated theories that combine aspects of both two-factor theory and cognitive expectancy ([Lovibond, Saunders, Weidemann, & Mitchell, 2008](#)). Others have suggested that avoidance behavior (initially reflecting an $S^D \rightarrow R \rightarrow O$ association) can be overtrained and disconnected from the outcome ([LeDoux & Daw, 2018](#)), leading to *habit slips* like those discussed in [Chapter 5](#), where the $S^D \rightarrow R$ association has become so automated that it occurs whether or not the outcome is expected any more. Avoidance learning may therefore be a useful tool for studying the potentially complex interactions between classical conditioning, operant conditioning, and cognitive processing—as well as a reminder that many seemingly “simple” behaviors may actually reflect more than one type of learning.

Learned Helplessness

Back in the 1960s, psychologist Martin Seligman and his colleagues studied an avoidance task involving a large box with a low barrier that divided the box into two chambers ([Overmier & Seligman, 1967](#); [Seligman & Maier, 1967](#)). They placed a dog in one chamber and periodically delivered a series of electric shocks through the floor of the box. At first, dogs reacted to the shocks by running around the chamber; eventually, they learned that if they would jump over the barrier into the opposite chamber, the shock would stop ([Figure 10.5](#)). In other words, the dogs

could escape from the shock by making a crossing response. If shocks were always preceded by a warning signal, such as a tone, the dogs soon learned to cross into the opposite chamber as soon as the tone sounded, thus avoiding the shocks altogether.



Description

A warning buzzer placed next to the platform, rings. Electric shock is passed on the section of the platform where the dog stands. The dog puts a leg forward across the plank, in an attempt to escape.

Seligman and his colleagues wondered what would happen if, before a dog was first put in the avoidance box, the tone stimulus had already been explicitly paired with a shock from which the dogs could not escape, in a classical fear-conditioning paradigm:

CS (tone) → US (shock)

If the dog had already learned this CS–US association and knew that the tone signaled upcoming shock, would the dog be able to transfer this learning to the avoidance learning paradigm? Might the dog jump over the barrier the very first time it heard the tone in order to avoid being shocked?

What the dogs actually did surprised everyone. The first time they heard the tone, the dogs ran around for a few seconds—and then they lay down in the corner and whined. Even after repeated trials, the dogs never learned to cross to the other chamber and escape from the shock. Intrigued, the experimenters tried to encourage the animals to escape: removing the barrier wall, baiting the opposite chamber with food, even climbing into the far chamber themselves and calling to the dogs to cross over. Still, the dogs continued to huddle in the corner, enduring the shock.

Seligman concluded that the prior exposure to an inescapable shock (during the classical-conditioning phase) had taught the animals that they were helpless to escape *any* shock—even in the operant-learning phase. Seligman named this phenomenon **learned helplessness**, meaning that exposure to an uncontrollable punisher teaches an expectation that responses are ineffectual, which in turn reduces the motivation to attempt new avoidance responses. Learned helplessness has since been demonstrated in a variety of species, from cockroaches ([Brown & Stroup, 1988](#)) to rats ([Besson, Privat, Eschaliere, & Fialip, 1999](#)) to humans ([Hiroto, 1974](#); [Hiroto & Seligman, 1974](#)).

learned helplessness

A phenomenon in which exposure to an uncontrollable punisher teaches an expectation that responses are ineffectual, which in turn reduces the motivation to attempt new avoidance responses.

Understanding this phenomenon may provide clues for how to treat or protect against **depression**, a psychiatric condition characterized by sadness and a general loss of initiative and activity (see “[Learning and Memory in Everyday Life](#)”). Seligman himself went on to study what he called “positive psychology,” arguing that just as negative emotional responses such as fear and unhappiness can be learned, so can positive emotional responses such as happiness and hope for the future ([Seligman, 1991](#); [Seligman, Steen, Park, & Peterson, 2005](#)).

depression

A psychiatric condition that involves sadness as well as a general loss of initiative and activity.

LEARNING AND MEMORY IN EVERYDAY LIFE

“Immunizing” Against Learned Helplessness

Learned helplessness was originally discovered and studied under highly controlled laboratory conditions. But researcher Martin Seligman suggested that learned helplessness might be an important component of human depression ([Seligman, 1975](#)). People suffering from depression spend a lot of time sitting around or sleeping, sometimes skipping work or school because they lack the energy to leave home. Some depression appears to be triggered by external problems, but affected

individuals often feel unable to do anything to change the conditions that are making them feel depressed. This somewhat resembles the behavior of Seligman's dogs, who helplessly endured pain rather than explore ways to escape from or avoid it. The idea that learned helplessness may at least partially underlie human depression is supported by the finding that antidepressant drugs, which alleviate depression in humans, can also eliminate learned helplessness in rats previously exposed to inescapable shock ([Besson et al., 1999](#)).

If learned helplessness does play a role in human depression, then perhaps the same behavioral techniques that help animals overcome learned helplessness could also benefit depressed patients. For example, Seligman found that if he first trained his animals to escape the shock, then exposed them to inescapable shock, and then tested them again, the animals continued to make escape responses ([Seligman, Rosellini, & Kozak, 1975](#)). Apparently, the earlier learning that they could escape shocks "immunized" the animals against learned helplessness when they were later exposed to inescapable shock. Perhaps humans could be "immunized" against depression in the same way: if humans are exposed early in life to adversities that they can overcome, perhaps this early training could protect them against learned helplessness when confronted with more difficult challenges later in life.

The same insight could apply to educational methods. Some children attribute their academic failures to inescapable conditions: "I'm bad at math" or "I'm not as smart as the other kids." Confronted with a new math problem, such a child might not even bother to try—just like Seligman's helpless dogs. Maybe this attitude could be reversed or lessened by a training procedure that starts with problems the child can solve and intermixes progressively harder ones, gradually teaching the child a way to "escape" from the cycle of failure by exposure to challenges that he can master.

Memory for Emotional Events

So far, you've read about conditioned emotional responses to stimuli that predict upcoming threat and also about learning to escape or avoid such events. But there is another important way in which emotion interacts with learning and memory: strong emotions can affect the storage and retrieval of episodic memories.

Let's turn again to the morning of September 11, 2001. Most Americans who were old enough to understand what was happening experienced strong emotional responses, ranging from sadness at the deaths, to anger at the terrorists, to fear that further attacks would follow. And many report that they still have vivid, detailed memories of that day. This phenomenon is not limited to negative emotions. Think back to the happiest day of your life. You can probably describe that day in a comparable level of detail.

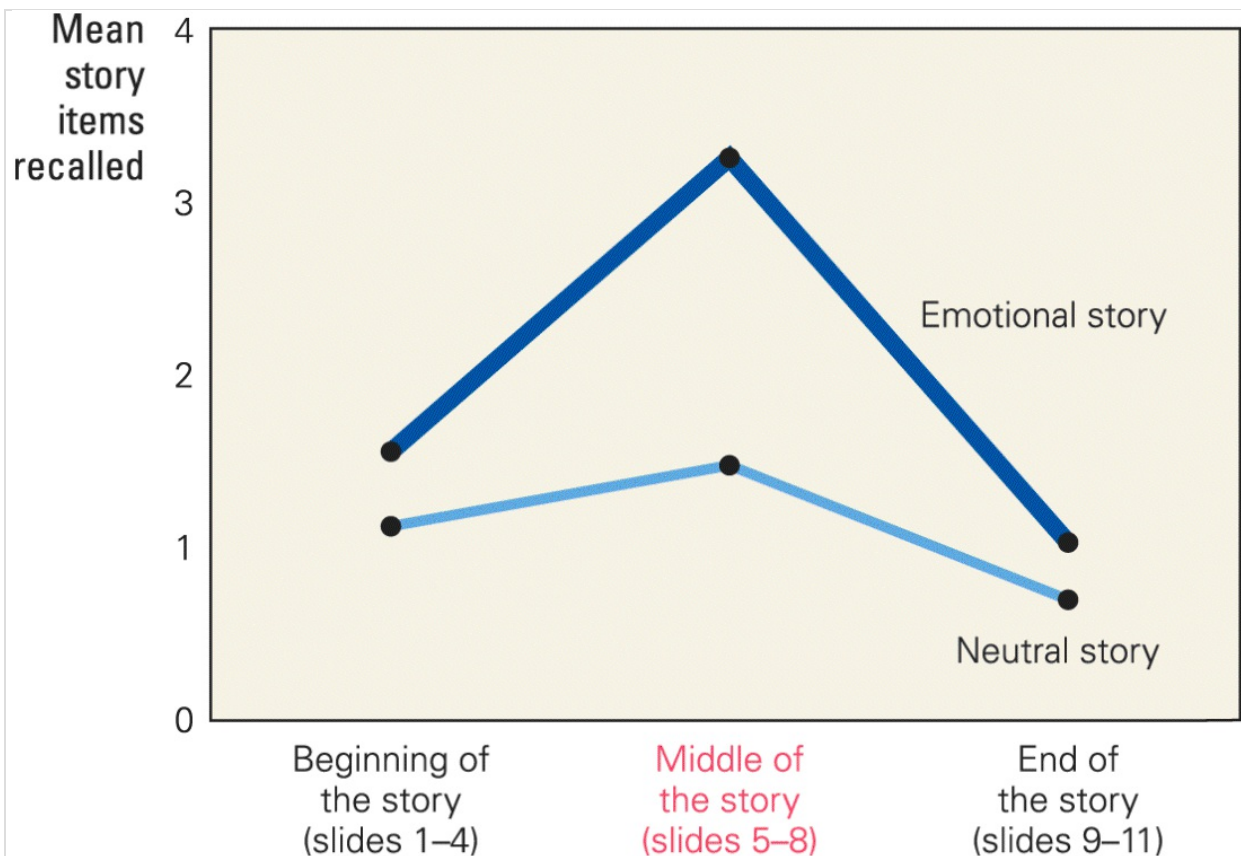
One reason we often have strong memories for episodes of intense emotion (fear and anger but also happiness and surprise) is that these tend to be important memories that we rehearse frequently, reviewing them mentally and talking about them with others ([Heuer & Reisberg,](#)

[1992](#)). So, for example, in the days following the 9/11 attacks, people discussed the situation with friends and family members and watched intensive TV coverage—and each time, their memories for the original event were retrieved, rehearsed, and strengthened. (As [Chapter 7](#) points out, rehearsal strengthens memory.) However, the effect of emotion goes beyond rehearsal: strong emotions can actually affect the probability that an episodic memory is encoded in the first place.

Emotion and Encoding of Memories

Researchers can study the effects of emotional content on the strength and specificity of memories by creating emotional experiences in the laboratory and then testing for memories of them. In a classic set of experiments, one group of participants saw a slide show accompanied by a highly emotional story. The beginning of the story described a boy who went on an outing with his mother; in the dramatic middle part, the boy was involved in a traumatic accident and was rushed to surgery; finally, at the end of the story, his mother arranged to take him home ([Cahill, Babinsky, Markowitsch, & McGaugh, 1995](#); [Cahill & McGaugh, 1995](#); [Heuer & Reisberg, 1990](#)). A second group of participants saw the same slide show, with the same narrative accompanying the beginning of the story (mother takes boy out) and end of the story (mother takes boy home). But for this second group, the middle part of the narrative was changed: these participants saw the same surgery-related photos, but they were told that the boy was observing the hospital's disaster drill, during which hospital workers were practicing their skills on volunteers made up to look like trauma victims.

Two weeks later, participants were shown the pictures again and asked to recall the accompanying narrative. Those who had heard the emotional accident story remembered the dramatic middle events very well; their memories for the comparatively unemotional beginning and end parts were somewhat worse ([Figure 10.6](#)). In contrast, participants who had heard the less exciting story about a disaster drill recalled fewer events, particularly from the middle ([Cahill, Uncapher, Kilpatrick, Alkire, & Turner, 2004](#)). Apparently, even though both groups of participants saw the same pictures, those for whom the story was emotionally arousing were better able to encode the details and recall them later ([Cahill et al., 1995](#)).



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FIGURE 10.6 Memory for emotionally arousing material Participants who heard the emotional middle part of the story (in which the boy was injured) were later able to recall more details of that middle part than of the less dramatic beginning and end of the story. By comparison, participants for whom the middle part of the story was emotionally neutral (the boy merely observed a hospital drill) showed no enhanced memory for the middle of the story.

Research from [Cahill et al., 1995](#).

Description

The readings on the vertical axis range from 0 to 4 in increments of 1 and the labels on the horizontal axis read as follows. Beginning of the story (slides 1 to 4), Middle of the story (slides 5 to 8), and End of the story (slides 9 to 11). The graph has two curves representing the neutral story and the emotional story. The data in the graph is as follows.

Neutral story: Beginning of the story (slides 1 to 4) - mean number of items recalled is 1.2, Middle of the story (slides 5 to 8)-mean number of items recalled is 1.3, End of the story (slides 9 to 11)-mean number of items recalled is 0.7.

Emotional story: Beginning of the story (slides 1 to 4) - mean number of items recalled is 1.5, Middle of the story (slides 5 to 8)-mean number of items recalled is 3.3, End of the story (slides 9 to 11)-mean number of items recalled is 1.

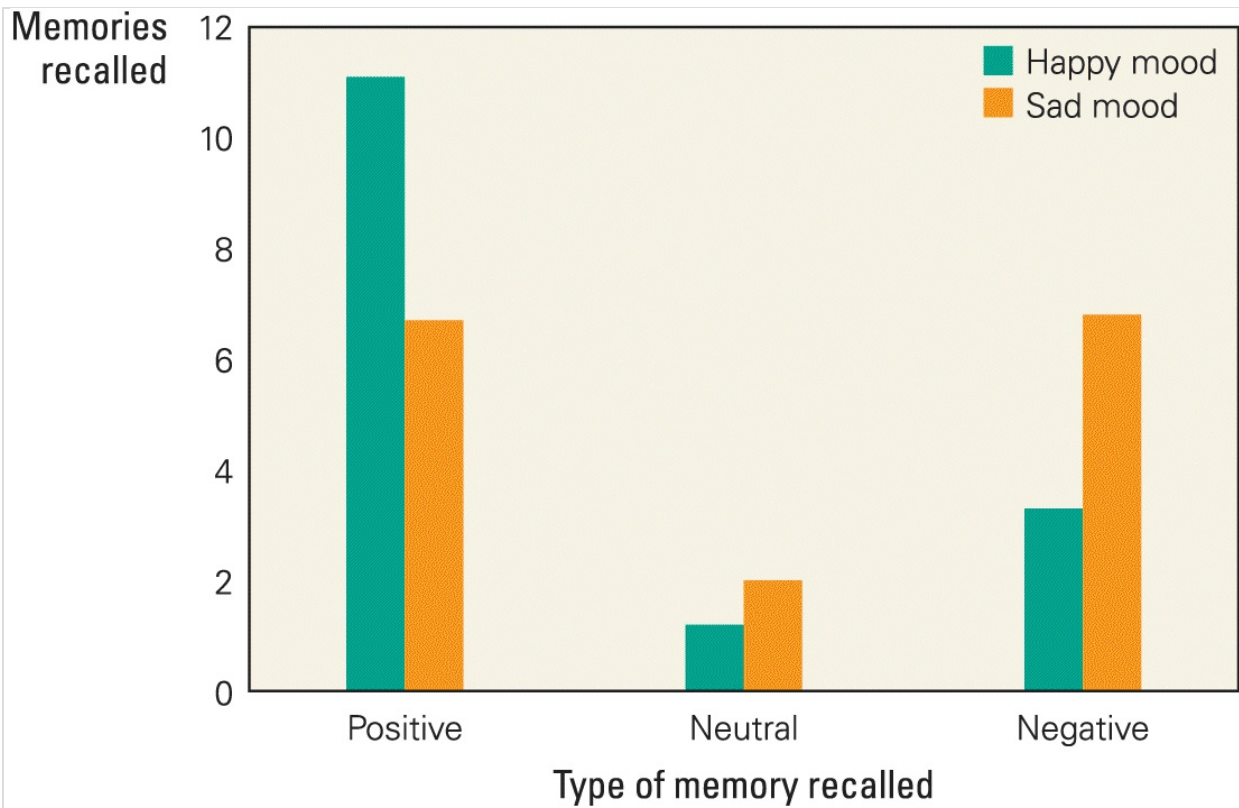
The strong encoding and persistence of emotionally charged memories is one reason advertisers

often use arousing images in their commercials. If they can evoke pleasurable emotion such as excitement or humor, there is a better chance you will remember the commercial and, perhaps, buy the product later. Some advertisers take this attempt at emotional manipulation a step further, using overtly sexual or provocative images, even to pitch comparatively nonerotic items such as deodorant or hamburgers. The hope is that viewers will experience sexual arousal while viewing the ad, and misinterpret this arousal as an attraction to the product—just like the participants in the “High Bridge” study who misinterpreted their arousal as an attraction to the researcher.

Emotion and Retrieval of Memories

Emotion not only affects how memories are encoded (their strength and specificity); it also affects their retrieval. [Chapter 7](#) discusses the *encoding specificity effect*, which states that chances of recall are increased if the cues and context available at the time of testing are similar to the cues and context that were available at the time the material was learned or encoded. Our emotions are part of our internal context, so the encoding specificity effect applies to emotions as well: it is easier to retrieve memories that match our current mood or emotional state.

This effect is termed **mood congruency of memory**. For example, in one study, students first listened to music and reported whether it made them feel happy or sad. They then saw a list of words (SHIP, STREET, etc.) and were asked to recall an autobiographical memory associated with each word. Students who had reported being in a happy mood tended to generate mostly positive memories and relatively few neutral or negative memories, as shown by the green bars in [Figure 10.7](#) (Eich, Macaulay, & Ryan, 1994). Students who had reported being in a sad mood (orange bars in [Figure 10.7](#)) recalled fewer positive memories and more negative memories than the happy students. Few students in either mood generated many neutral memories—consistent with the general principle that our strongest memories tend to be associated with strong emotions, whether those emotions are positive or negative.



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FIGURE 10.7 Mood congruency of memory People in a happy mood tend to recall more positive than negative or neutral autobiographical memories; by contrast, people in a sad mood recall significantly fewer positive and more negative memories.

Data from [Eich & Macaulay, 2000](#).

Description

The readings on the vertical axis range from 0 to 12 in increments of 2 and the types of memory recalled on the horizontal axis are labeled, positive, neutral, and negative. The data in the graph is as follows.

Positive: happy mood-memories recalled is 11, sad mood-memories recalled is 7.

Neutral: happy mood-memories recalled is 1.5, sad mood-memories recalled is 2.

Negative: happy mood-memories recalled is 3.5, sad mood-memories recalled is 7.

All the above data is approximate.

mood congruency of memory

The principle that it is easiest to retrieve memories that match our current mood or emotional state.

Mood-congruent memory effects occur in real-life contexts, too. For example, patients suffering

with clinical depression, which includes feelings of extreme sadness and hopelessness, are more likely to recall sad or unpleasant events than pleasant memories ([Clark & Teasdale, 1982](#); [Fogarty & Hemsley, 1983](#)). This can lead to a vicious cycle in which the recollection of sad memories makes the patient feel even more depressed and hopeless.

Why might mood influence recall? One reason is that an emotion entails physiological responses, overt behaviors, and subjective feelings, all of which can be incorporated into the memory just like other contextual cues. As discussed in [Chapter 7](#), one of the factors influencing our ability to retrieve a memory is the number of cues available to guide retrieval. From this perspective, a strong mood or emotion is simply one kind of memory cue; as the number of cues available at the time of recall increases, the more likely we are to retrieve the memory.

Flashbulb Memories

You have just read how our everyday emotional highs and lows can strengthen memories. Extreme emotions can result in memories that appear to have exceptional strength and durability. Well-studied examples of this phenomenon are cases in which events occurred that evoked strong emotions in most members of society—for instance, the assassinations of public figures such as President John F. Kennedy or Martin Luther King, Jr., or natural disasters such as devastating hurricanes or floods. The resulting memories (like the ones many Americans retain nearly two decades after the 9/11 terrorist attacks) are called **flashbulb memories** because they form quickly, as if the brain were taking “flash photographs” to preserve the incident forever in searing detail ([Brown & Kulik, 1977/1982](#)). We may remember these events vividly, recollecting not just where we heard the news but even things like the smell of the room, the sound of a clock ticking in the background, and the color and texture of the clothes we were wearing. By contrast, memory for such details on other, ordinary days is generally much less detailed ([Paradis, Solomon, Florer, & Thompson, 2004](#)).

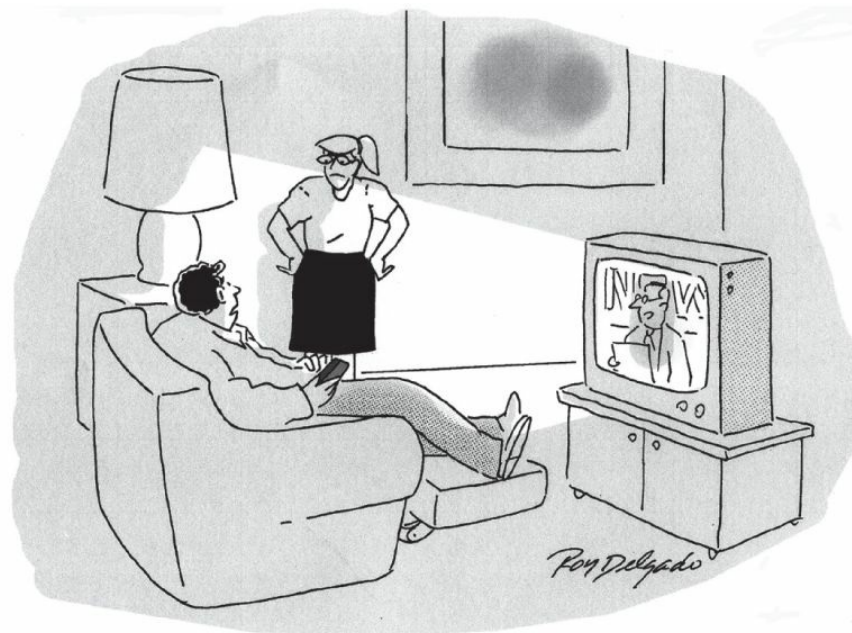
flashbulb memory

A memory formed under conditions of extreme emotions that seems especially vivid and long-lasting.

People generally report being very confident about the accuracy of their flashbulb memories. However, it appears that flashbulb memories are no more objectively accurate than other episodic memories ([Hirst & Phelps, 2016](#)). In terms of reliability, flashbulb memories are merely episodic memories that are experienced with great vividness and confidence ([Sharot, Martorella,](#)

[Delgado, & Phelps, 2007](#))—but not necessarily with greater accuracy than other memories.

For example, on September 12, 2001, the day after the 9/11 attacks, researchers Jennifer Talarico and David Rubin contacted 54 Duke University students and asked a series of open-ended questions about how the students had heard the news ([Talarico & Rubin, 2003](#)). On average, students remembered about 12 details, such as where they had been, who was with them at the time, and so on. Talarico and Rubin then contacted the students again, either 1, 6, or 32 weeks later. While most students reported that the memories were still vivid, the memories did change over time. Students contacted a week after the event remembered only about 10 of the details they had previously reported, and students contacted 8 months after the event remembered only about 7 details. At the same time, inconsistent details crept into the reports. For example, on September 12, 2001, one student reported hearing the news from a friend; by May 2002, that same student “remembered” having heard the news on television. The distortions then tended to persist, such that participants who reported an incorrect detail when tested a year after the events might report the same incorrect detail 10 years later ([Hirst et al., 2015](#)).



**" Sure I remember our 20th wedding anniversary - that was
the year Denver beat Atlanta, 31-19 in the Super Bowl ! "**

Roy Delgado/www.CartoonStock.com

What features of an event make it likely to produce flashbulb memories?

Description

The man is seated on a couch. Text at the bottom reads, sure I remember our twentieth wedding anniversary. That was the year Denver beat Atlanta, 31 to 19 in the super bowl!

A similar pattern of distortion over time has been demonstrated for memories of other notable events ([Neisser & Harsch, 1992](#); [Schmolck, Buffalo, & Squire, 2000](#)). The bottom line from all these studies is that flashbulb memories are long-lasting, vivid, and largely accurate—but they are not perfect photographic records: they can be incomplete and can contain inaccurate details.

Why should such errors creep into our memories? One possible cause is source monitoring errors, as discussed in [Chapter 7](#), such as forgetting that a friend told you the news and instead “remembering” that you first heard it on the television or radio ([Greenberg, 2004](#)). This is perhaps an understandable mistake, given our cultural tendency to watch and rewatch news footage following a national tragedy; the repetition and visual impact of news coverage may swamp our memories of how we really heard the news ([Hirst & Phelps, 2016](#)). Another factor is that memories of particularly important events are continuously pondered, rehearsed, and discussed. Each time, we are liable, quite unconsciously, to fill any little gaps in our memory with details that seem to fit the context. Later, we remember those inserted details as part of the original event. As discussed in [Chapter 7](#), this is one way that false memories can form. The result is that although many remembered details may be quite correct, others—that seem equally vivid—may be entirely wrong.

In fact, numerous studies have now documented that, while emotion often enhances memory for key events, this benefit does not always extend to background details ([Kensinger, Piguet, Krendl, & Corkin, 2005](#); [Kim, Vossel, & Gamer, 2013](#); [Waring & Kensinger, 2011](#)). For example, in one study, subjects saw photos in which emotional stimuli (e.g., a snake) or neutral stimuli (e.g., a chipmunk) were placed in the context of a neutral background scene (e.g., a riverbank). Later, participants were given a recognition test in which stimuli and scenes were presented separately. Unsurprisingly, memory was typically better for the emotional stimuli (snake) than for the neutral stimuli (chipmunk)—but memory was worse for the background scenes that had been presented with an emotional stimulus than for scenes presented with a neutral stimulus ([Waring & Kensinger, 2009](#)). In effect, there is a “trade-off” in which the strong memory for the central emotional content comes at the cost of weaker memory for surrounding details.

So, think back to some memorable occasion during which you heard important, emotionally arousing news, such as a school shooting or the death of someone close to you. Where were you when you got the news? What were you doing at the time? Who was with you? It may be instructive to ask those people what they remember about the day and to see whether their memories match yours. Chances are, you will be right about many of the important facts—after all, strong emotions do encourage strong memories—but at least some of the smaller details that you remember quite vividly may indeed turn out to be false memories (see “[Learning and Memory in Everyday Life](#)”).

LEARNING AND MEMORY IN EVERYDAY LIFE

Truth or Consequences

In February 2015, Brian Williams, then the anchorman of NBC’s prestigious *Nightly News*, became the center of controversy related to his descriptions of being in a helicopter forced down by enemy fire in Iraq in 2003. In fact, what appears to have happened is that the helicopter ahead of him was hit and made an emergency landing; Williams himself was on another helicopter following some distance behind ([Somaia, 2015](#)). In the wake of the controversy, NBC suspended Williams, who stated in a public apology that he had misremembered the event.

How could someone possibly misremember such an important event? In his initial on-air report of the incident in 2003, Williams said that the helicopter he was flying in was ordered to land quickly and that the helicopter ahead had been “almost blown out of the sky.” By 2007, Williams was telling a version of the story in which his own helicopter had come under fire, and in a 2013 TV interview, Williams described how his helicopter had been shot down.

Some accused Williams of exaggerating or misrepresenting the incident to embellish his public image. Memory researchers, in general, were kinder, noting that, while it’s possible Williams simply lied, it’s also quite plausible that, in telling and retelling the story over the years, distortions crept in, and details became confused ([Bello, 2015](#)). In this case, the terror of hearing that the lead helicopter had been hit, and the subsequent confusion of an unscheduled landing in dangerous territory, may have gradually coalesced into a false memory that it was Williams’s own helicopter that had been fired upon. The more Williams talked about it, on TV and in his personal life, the stronger the false memory may have become.

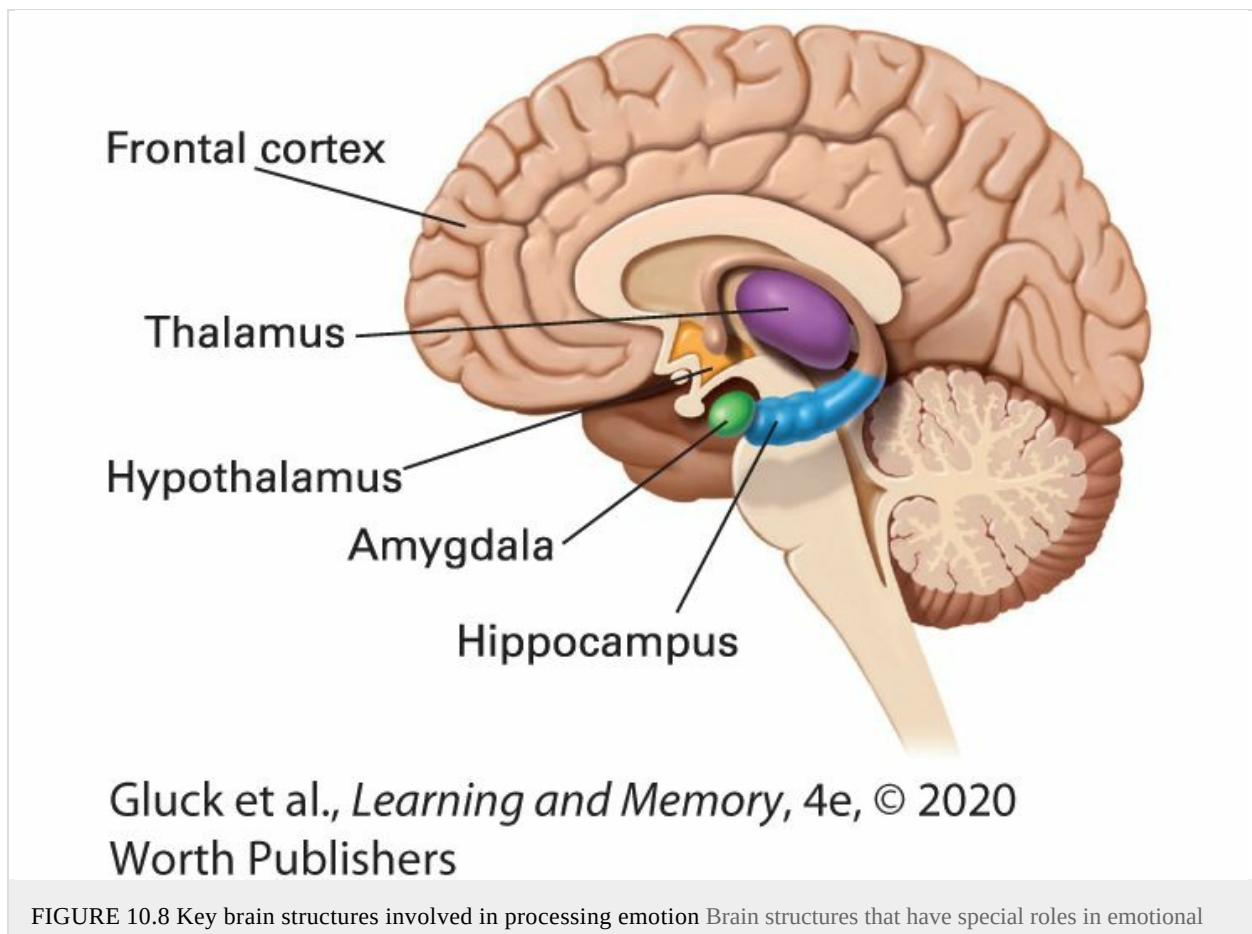
In 2008, then-Senator Hillary Clinton came under similar scrutiny for her repeated description of running off a plane under sniper fire during a 1996 trip to Bosnia; in fact, news footage of the event shows no evidence of sniper fire while Clinton and the other passengers emerge smiling from the plane ([Harnden, 2008](#)). As with Williams, it is possible that Clinton’s episodic memory may have become confused with another, similar incident where there was actual gunfire, or it might simply reflect that, at the time, she was sincerely worried about the possibility of snipers. Repeated re-imagining and retelling of the story may have led to distortion and confusion of details until she really believed she had been exposed to enemy fire that day.

For a political figure, the cost of being caught in a misstatement is a chance for the opposition to level accusations of dishonesty. For a journalist, the cost can be a loss of credibility as an unbiased presenter of the news. For the rest of us, episodes like these serve as reminders that memory is malleable and fallible and that telling a tale from memory is no substitute for checking the facts.

- Emotions have three components: physiological responses, overt behavioral responses, and conscious feelings.
- The major emotions, such as happiness, fear, and anger, seem to be universal, although the overt display of these emotions may be influenced by culture.
- Arousal, also called the fight-or-flight response, is the body's way of preparing to face or run away from a threat. Energy is diverted toward the brain, lungs, and legs and away from other systems such as the digestive and immune systems.
- The James–Lange theory of emotion and other somatic theories of emotion propose that bodily responses (e.g., physiological responses and overt behaviors) cause our conscious feelings of emotion. The Cannon–Bard theory proposes that bodily responses and conscious feelings of emotion are evoked simultaneously and independently. The two-factor theory of emotion posits that conscious feelings of emotion are produced by an interaction between bodily responses and our cognitive appraisal of the situation.
- While we cannot be sure whether nonhuman animals have conscious emotional feelings, their physiological and behavioral responses to emotional stimuli are often very similar to those in humans.
- Conditioned emotional learning is a form of classical conditioning in which a CS (e.g., a tone) that has been paired with an emotion-evoking US (e.g., an electric shock) evokes a CR (e.g., a freezing response or an increased heartbeat).
- Conditioned escape learning involves operant conditioning: a stimulus S^D (e.g., an electric shock) can evoke a motor response R (e.g., jumping over a barrier) to produce an outcome O (e.g., escape from shock).
- Avoidance learning, which means learning that a particular response results in avoidance of an aversive event, may involve both classical and operant conditioning; in addition, cognitive expectancies may influence the organism's choice between various possible responses.
- Learned helplessness occurs when exposure to an inescapable punisher impairs an organism's learning to escape or avoid future punishment.
- Emotions can increase the strength of memory encoding and the duration of memory storage. Emotion can also affect retrieval: we are more likely to retrieve memories that fit our current mood.
- Flashbulb memories are vivid and long-lasting, but they are not always completely accurate.

10.2 Brain Substrates

From the earliest days of brain science, scientists have tried to understand how the brain gives rise to emotion. In 1937, anatomist James Papez made one of the most influential attempts to locate the brain substrates of emotion. (Papez is pronounced “papes,” to rhyme with “grapes.”) Based on the information available to him at the time from patients with different types of brain damage, Papez concluded that several brain areas are important for emotions, including the *hippocampus*, which is vital for episodic memory formation; the *thalamus*, by which most sensory information enters the brain; and the *hypothalamus*, which helps regulate the body’s response to emotion. Papez proposed that these brain regions operate in a loop, later named the *Papez circuit*, that he described as the central processing pathway for emotion ([Papez, 1937](#)). A later modification of this idea, called the *limbic system*, identified the brain substrates of emotion as including the Papez circuit as well as the *amygdala*, parts of the frontal cortex, and other cortical and subcortical structures ([MacLean, 1949](#)). Some of these structures are shown in [Figure 10.8](#).



learning include the amygdala, hippocampus, thalamus, and hypothalamus. The frontal cortex also plays a key role in interpreting the context and monitoring the display of emotion.

Description

The parts labeled are as follows. Frontal cortex at the anterior region, thalamus and the hypothalamus at the center, Amygdala underneath the hypothalamus and Hippocampus at the rear side of the Amygdala.

Papez's proposed circuit is important historically because it was one of the first systematic attempts to understand the brain substrates of emotion. Remarkably, many of the presumed pathways connecting the structures in the Papez circuit were not yet known to exist; Papez merely deduced that they must be there. Almost all of them have since been discovered in the brain. We now know, however, that there is no specialized "emotion circuit." Each emotion activates many different brain regions. One report combined data from 55 studies of emotion that had used neuroimaging techniques such as fMRI to determine which brain areas are activated by the emotions happiness, sadness, disgust, fear, and anger ([Phan, Wagner, Taylor, & Liberzon, 2002](#)). The report concluded that each emotion activated many different brain regions, but no single brain region was activated by all the different emotions. Thus, emotion appears to be a function of the brain as a whole rather than arising from special circuits dedicated to individual emotions. All the same, some parts of the brain do appear to be particularly important for emotional learning. First and foremost among these is the amygdala.

Emotional Learning in the Brain

As shown in [Figure 10.8](#), the **amygdala** is a small almond-shaped structure that lies at the anterior tip of the hippocampus. (The word *amygdala* comes from the Greek word for "almond.") Just as the brain has one hippocampus in each hemisphere, it also has one amygdala in each hemisphere, at the tip of each hippocampus; but for convenience, most researchers refer to "the amygdala," just as they refer to "the hippocampus."

amygdala

A collection of brain nuclei that lie at the anterior tip of each hippocampus; critical for learning and expressing emotional responses as well as mediating the emotional modulation of memory formation.

The amygdala is a collection of more than 10 separate subregions, or *nuclei*, all of which have different input and output pathways. [Figure 10.9](#) depicts two key nuclei within the amygdala.

(Many other nuclei and pathways exist but are not shown in [Figure 10.9](#), for simplicity.) The *basolateral amygdala* (which itself consists of several subnuclei) is a primary entry point for sensory information into the amygdala; this sensory information arrives directly from the thalamus and also indirectly from the thalamus by way of the cortex. The basolateral amygdala sends axonal projections to the *central nucleus*, which in turn projects out of the amygdala to the autonomic nervous system (ANS), driving physiological arousal and release of stress hormones. The central nucleus also projects out to areas of the brain that produce motor responses, including behaviors such as freezing and startle. The amygdala is thus critical in both learning and expressing emotional responses. Let's look at these functions in more detail.

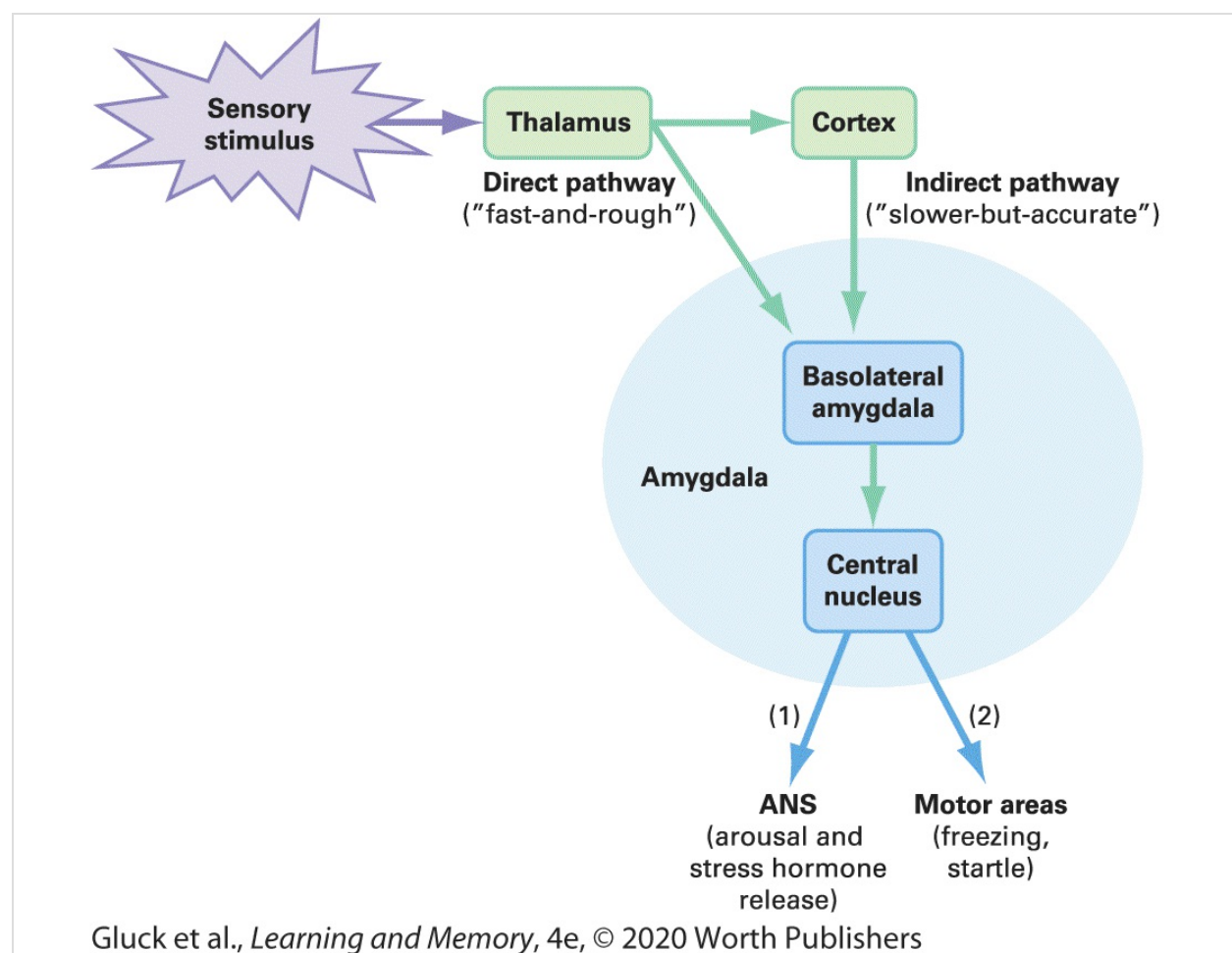


FIGURE 10.9 Some key nuclei of the amygdala and their major connections Fibers carrying sensory information from the thalamus and cortex project to the basolateral amygdala, which in turn sends projections to the central nucleus of the amygdala. The central nucleus sends projections (1) to the autonomic nervous system (ANS), driving expression of physiological responses, such as arousal and release of stress hormones; it also sends projections (2) to motor areas of the brain that produce the behavioral components of an emotional response, such as freezing and startle.

Data from [LeDoux, 2000](#).

Description

The information in the flowchart is as follows.

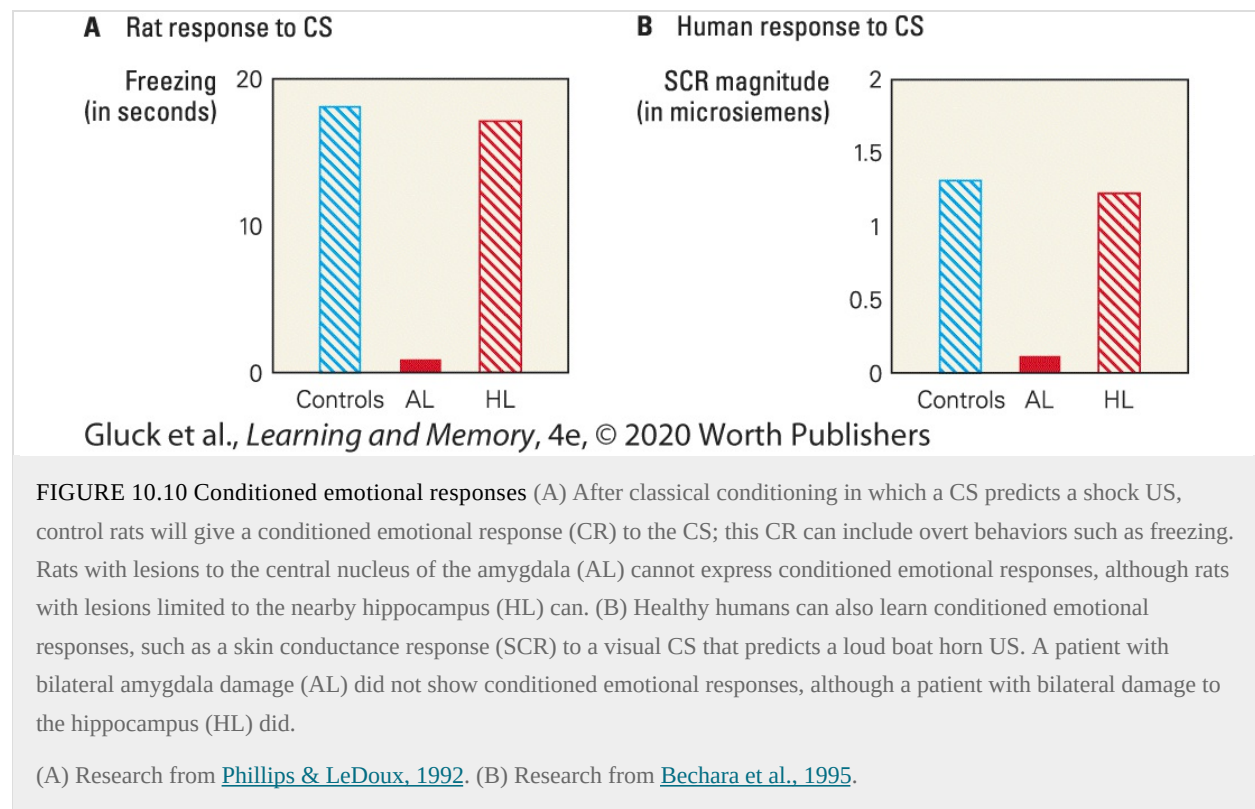
Sensory stimulus leads to the thalamus, which leads to the cortex. A direct pathway (fast and rough) exists between thalamus and Basolateral amygdala. An indirect pathway (slower but accurate) exists between the cortex and the Basolateral amygdala. Basolateral amygdala leads to the central nucleus of Amygdala. The central nucleus leads to: 1. A N S (arousal and stress hormone release) and 2. Motor areas (freezing, startle). The Basolateral amygdala and the central nucleus of the Amygdala are marked within a shaded circle.

The Amygdala and Expression of Emotional Responses

Because the amygdala projects out to the ANS and to motor areas of the brain, amygdala activity can trigger both physiological arousal and behavioral responses. In the research lab, electrical stimulation of the amygdala can produce dramatic emotional displays. For example, stimulating the amygdala of a predator, such as a cat, can cause a species-typical defensive reaction, including lowering of the head, flattening of the ears, piloerection, and growling or hissing ([Roldan, Alvarez-Pelaez, & Fernandez de Molina, 1974](#)). In a prey animal, such as a rabbit, amygdala stimulation causes a different species-typical defensive reaction, including freezing and a lowered heart rate ([Kapp, Gallagher, Underwood, McNall, & Whitehorn, 1981](#)). Stimulating the amygdala in humans does not produce such dramatic results. Humans given amygdala stimulation may report subjective feelings of mild positive or negative emotion but generally do not exhibit strong behavioral and physiological responses such as occur in cats and rabbits ([Bancaud, Brunet-Bourgin, Chauvel, & Halgren, 1994](#); [Halgren, 1982](#)).

Why might stimulation of the amygdala cause such a dramatic emotional response in some animals but only mild feelings of foreboding in humans? According to the two-factor theory of emotion (see [Figure 10.2C](#)), our conscious emotional feelings depend not only on our physiological responses but also on how we interpret the situation. In the relatively “safe” context of a lab environment, human participants receiving amygdala stimulation know they are in a controlled situation and that a full panic response is not warranted. On the other hand, in a truly menacing situation—such as a dark alley at night—amygdala activation would produce bodily responses that, combined with cognitive appraisal of the potential for danger, might elicit feelings of terror.

Given that the amygdala helps initiate the body's emotional responses, it is not too surprising that lesions of the central nucleus of the amygdala disrupt the ability to learn and display new emotional responses. For example, [Figure 10.3](#) illustrated conditioned emotional responding in which healthy rats, having been trained that a CS predicts a shock US, will give CRs that can include physiological changes (e.g., increased blood pressure) and overt behavioral responses (e.g., freezing). Outputs from the central nucleus of the amygdala drive these emotional responses. [Figure 10.10A](#) shows that rats with lesions to the central nucleus of the amygdala do not show a learned freezing response, although rats with damage limited to the nearby hippocampus can learn and express the emotional CR as effectively as control rats do ([Phillips & LeDoux, 1992](#)).



Description

Rat response to C S: the graph plots freezing time in seconds on the vertical axis ranging from 0 to 20 in increments of 10. The freezing time of the three groups read as follows. Controls- 18 seconds, A L- 2 seconds, and H L- 17 seconds.

Human response to C S: the graph plots S C R magnitude in microsiemens on the vertical axis ranging from 0 to 2 in increments of 0.5 The S C R magnitude for the three groups read as follows. Controls- 1.4 microsiemens, A L- 0.2 microsiemens, and H L- 1.3 microsiemens.

All the above data is approximate.

In humans, bilateral damage limited to the amygdala is rare ([Markowitsch et al., 1994](#)), but a few such cases have been identified and studied. These patients often show deficits in learning emotional responses. The *skin conductance response* (SCR), for instance, is a tiny but measurable change in the electrical conductivity of the human skin that occurs when people feel arousal. In healthy people, a loud sound, such as a 100-decibel boat horn (the US), produces a sharp increase in skin conductance (the UR). If this US is always preceded by a neutral stimulus, such as a colored shape, healthy participants learn that the CS predicts the US, and they subsequently produce an SCR to the CS alone ([Figure 10.10B](#)). In contrast, a patient with bilateral amygdala damage would exhibit arousal when startled by the boat horn US but could not learn a conditioned emotional response to the CS that predicted the US ([Bechara et al., 1995](#)). A different patient with damage that included the nearby hippocampus (but not the amygdala) could learn a conditioned emotional response as well as control participants.

These disruptions in conditioned emotional responses occur because the central nucleus provides the major outputs from the amygdala both to the ANS (output pathway 1 in [Figure 10.9](#)) and to motor centers that drive the freezing response (output pathway 2 in [Figure 10.9](#)). Lesion of the central nucleus therefore disrupts the ability to express a learned fear response. Some researchers believe that the conditioned emotional response is learned and stored in the basolateral amygdala complex, with the central nucleus behaving more like a simple waystation that signals other brain stations to turn on the fear responses ([Cahill, Weinberger, Roozendaal, & McGaugh, 1999](#); [Fanselow & LeDoux, 1999](#); [Maren, 1999](#); [McGaugh & Cahill, 2003](#)). According to this view, animals with lesions of the central nucleus might still be able to use their basolateral amygdala to *learn* about a fear-evoking CS; they would simply be unable to *express* this learning because the central amygdala outputs are disabled. Other researchers suggest that the central nucleus is not just a passive waystation but actively contributes to emotional learning, particularly when pleasant rather than unpleasant stimuli are involved ([Balleine & Kilcross, 2006](#); [Everitt, Cardinal, Parkinson, & Robbins, 2003](#)).

Two Pathways into the Amygdala: “Fast-and-Rough” Versus “Slower-but-Accurate”

As you saw in [Figure 10.9](#), the amygdala receives sensory input from two separate pathways

([Garcia, 2017](#)). The thalamus is the first site in the brain where most kinds of sensory input (visual signals from the retina, touch signals from the fingers, etc.) converge and are processed; for this reason, the thalamus is sometimes called the “sensory gateway to the brain.” From the thalamus, stimulus information travels directly to the amygdala and also to the cortex. As described in [Chapter 3](#), the cortex processes sensory information more fully, discriminating fine details and so on. Information from the cortex also travels down to the amygdala. Emotion researcher Joseph LeDoux proposed that both pathways—the direct path from the thalamus to the amygdala and the indirect path from the thalamus to the cortex to the amygdala—play important roles ([LeDoux, 1993, 1994](#)).

The direct path is faster—carrying information from the thalamus to the amygdala in about 12 milliseconds in rats—but it conveys less detail, just the bare outlines of stimulus information. The indirect pathway is slower—taking about 19 milliseconds for information from the thalamus to travel via cortex to the amygdala in rats—but the involvement of the cortex allows much finer discrimination of stimulus details. In LeDoux’s terms, the direct path is “fast and rough,” and the indirect path is “slower but accurate.” The faster, direct path allows us to react quickly, triggering an emotional response; the slower, more accurate path then provides extra information, allowing us to modulate the emotional response appropriately.

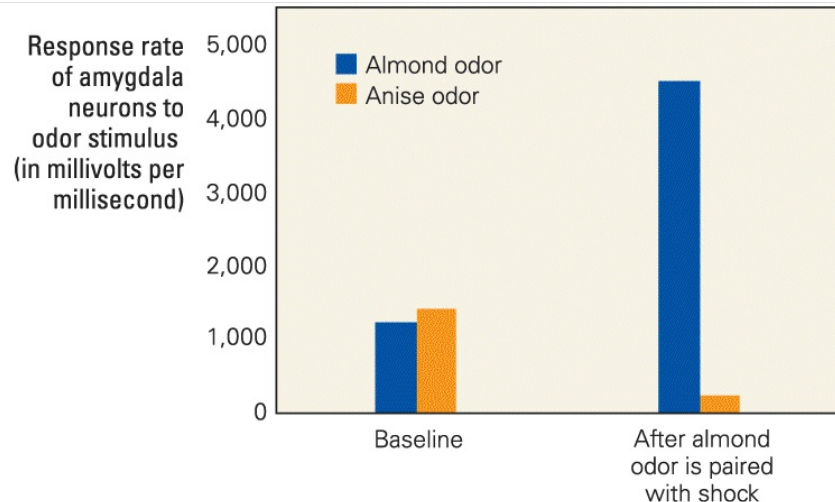
How might this dual pathway work? Suppose you are walking home alone at night, feeling a little nervous. A big, dark shape approaches. Information about the visual stimulus arrives at your thalamus, which shoots this information directly to the amygdala via the direct pathway shown in [Figure 10.9](#). The amygdala interprets this stimulus as possibly signaling danger and activates an immediate fear response (“Yikes!”), preparing you to fight or run away. A few milliseconds later, the signals from the thalamus reach your visual cortex, which processes the stimulus more fully and allows you to recognize the stimulus as nonthreatening: it’s only a friend walking toward you. This information (“No danger here”) reaches the amygdala via the indirect pathway in [Figure 10.9](#), shutting off the fear response. (It may take a few moments for stress hormone levels to decline enough so that your heart rate returns to normal and your goosebumps fade away.)

The cost of using the “fast-and-rough” direct pathway from thalamus to amygdala is that you have gotten yourself all worked up over nothing. On the other hand, the cost of overreacting to a

harmless stimulus is much less, in general, than the cost of failing to respond to a truly dangerous stimulus ([LeDoux, 2000](#); [LeDoux, Iwata, Cicchetti, & Reis, 1988](#)). If the approaching shape really had been a threat, the cost of waiting for your cortex to make a definitive identification could have been the difference between escape and injury. From an evolutionary perspective, animals with a genetic predisposition to freeze or dart for cover when they see movement in the bush (“Was that rustling in the bushes caused by a predator or just the wind? Better to play it safe and survive another day.”) are more likely to live long enough to produce offspring that share the same cautious instincts.

Where Emotional Responses Are Learned

Once an organism survives a brush with danger, the next challenge is to learn from it. Some of this learning probably takes place in the basolateral amygdala, where neural connections change as a result of experiencing a neutral CS paired with a fear-evoking US ([Maren, 1999](#)). For example, in one study, researchers implanted recording electrodes into the basolateral amygdala of rats to monitor the activity of about 100 neurons simultaneously ([Rosenkranz & Grace, 2002](#)). When the rats were first presented with various odors—say, almond and anise—the neurons showed a low baseline level of response to each ([Figure 10.11](#), baseline condition). The researchers then trained the rats that one of the odors—say, almond—always preceded a tail-shock US. Not surprisingly, the rats quickly learned to freeze in anticipation of a shock whenever they encountered the almond odor, although they showed no such fear response to the anise odor, which had not been paired with shock. [Figure 10.11](#) shows that, after training, the neurons in the basolateral amygdala responded strongly to the almond odor that had been paired with the US.



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FIGURE 10.11 The basolateral amygdala and conditioning Baseline responding of basolateral amygdala neurons of a rat is similar for two odors. If one of the odors (almond) is subsequently paired with a tail shock, the response to that odor increases strongly from the baseline value. This is exactly the pattern of neural responding that would be expected if the basolateral amygdala had encoded the associations between odor CS and shock US.

Data from [Rosenkranz & Grace, 2002](#).

Description

The data in the graph is as follows.

Baseline: the response rate is 1100 millivolts per millisecond for almond odor and 1200 millivolts per millisecond for anise odor.

After almond odor is paired with shock: the response rate is 4500 millivolts per second for almond odor and 100 millivolts per second for anise odor.

All the above data is approximate.

This is exactly the pattern you would expect to see if the learning to associate the CS and US were taking place in the basolateral amygdala. [Chapter 2](#) describes *long-term potentiation* (LTP), which is thought to be one mechanism of learning in the brain; in essence, “neurons that fire together wire together.” An interpretation of [Figure 10.11](#) on that basis would assume that the almond odor activates sensory neurons in the rat’s nose, which project (directly and also by way of sensory cortex) to neurons in the basolateral amygdala. These amygdala neurons would be strongly activated when the animal receives a shock US. Initially, the odor triggers little activity in these amygdala neurons (the “baseline” condition in [Figure 10.11](#)). But if the almond odor is

paired with the shock, the almond odor arrives just as the amygdala neurons are activated by shock, and since “neurons that fire together wire together,” the synapse between almond signals and amygdala neurons will be strengthened. The end result is that when the almond odor is presented again, it will now trigger more activity in the basolateral amygdala neurons (the “after” condition in [Figure 10.11](#)). No such strengthening occurs in pathways carrying information about anise or other odors that have not been paired with shock.

Further evidence of the role of the amygdala in fear conditioning comes from [optogenetics](#), a technique in which researchers inject living animals with a virus that will insert new genes into specific cells, causing those cells to become sensitive to light ([Deisseroth, 2010](#)). If the infected cells are neurons, researchers can then use light stimulation to selectively activate and deactivate those neurons, rather like flipping a switch to turn an electrical appliance on and off. One use of this technique has been to examine whether activity in some neurons actually causes changes in other parts of the brain.

optogenetics

A technique for causing specific neurons or other cells to become sensitive to light, after which researchers can use light stimulation to turn those cells “on” and “off” at will.

In one study using optogenetics, direct activation of an animal’s amygdala neurons triggered a freezing response. After an auditory CS was paired with this optogenetic activation a few times, the CS by itself evoked conditioned freezing ([Johansen et al., 2010](#)). In other words, the animals learned a conditioned fear response to the CS—even though they had never experienced an actual pairing of the CS with a shock. Other studies have replaced the tone CS with optogenetic stimulation of the auditory pathway into the amygdala and showed that when this stimulation is paired with a shock US, the animals will learn to freeze to the optogenetic stimulation ([Nabavi et al., 2014](#)). Together, these experiments show that the amygdala is the place where the CS and US information converge and where the CS–US association is formed.

Where Context Is Learned

In classical conditioning, a normal rat or human learns not only about the CS and the US but also about the context in which the US is likely to occur. Thus, after CS–US training in a particular chamber, the rat may show a conditioned emotional response (freezing) the next time it is put into that chamber—before any CS (or US) is presented. In fact, a healthy animal’s response to

the context may be almost as strong as its response to the CS! As discussed in [Chapter 6](#), contextual learning often depends on the hippocampus. This is true for emotional learning, such as fear conditioning, as well as for the types of learning explored in [Chapter 6](#). The hippocampus projects to the amygdala, both directly and indirectly (via the cortex and other brain structures), allowing contextual information from the hippocampus to help the amygdala trigger emotional responses.

For example, consider rats that have learned a freezing response to a tone CS that predicted an aversive shock US. [Figure 10.10A](#) showed that such CS–US learning is not disrupted in rats with hippocampal lesions, but it is devastated in rats with amygdala lesions ([LeDoux 1994](#)). [Figure 10.12A](#) shows what happened when the same rats were placed back in the conditioning context without any CS or US present. The control animals gave a strong freezing response—as if recognizing that they were back in a dangerous place. The rats with amygdala lesions gave no freezing response—probably because the lesions disrupted the output pathway from the amygdala to the motor areas that would generate the freezing response. But the rats with hippocampal lesions also showed very little freezing, indicating that they had learned little about the context ([Phillips & LeDoux, 1992](#)).

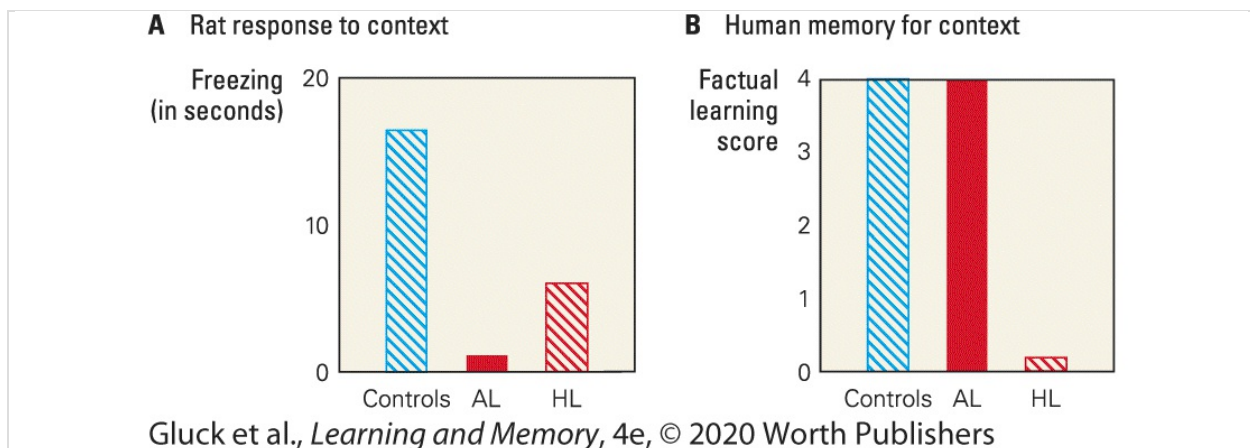


FIGURE 10.12 Conditioned contextual learning (A) During conditioning of an emotional response such as freezing in response to a CS that signals shock, control rats also learn a response (freezing) to the context where shock was experienced. This contextual learning is impaired by either amygdala lesion (AL) or hippocampal lesion (HL). (B) Healthy humans can report contextual information about a skin conductance conditioning experiment in which they are participating. AL spares this contextual memory, but HL greatly impairs it. These data suggest a dissociation between conditioned emotional responding to the CS (which depends on the amygdala) and contextual learning (which depends on the hippocampus).

(A) Research from [Phillips & LeDoux, 1992](#). (B) Research from [Bechara et al., 1995](#).

Description

Rat response to context: The graph shows freezing time in seconds on the vertical axis for three groups: controls, A L, and H L. The freezing time is as follows. Controls-16 milliseconds, A L-2 milliseconds, H L-5 milliseconds.

Human memory for context: The graph shows factual learning score for three groups: controls, A L, and H L. The factual learning score is as follows. Controls-4, A L-4, and H L-0.2.

All the above data is approximate.

A similar interplay occurs between hippocampus and amygdala in humans monitored for the skin conductance response. [Figure 10.10B](#) showed that healthy humans give a skin conductance response (SCR) to a CS that has been paired with a US (loud boat horn). After conditioning, the participants were asked what they remembered about the experiment ([Bechara et al., 1995](#)). The healthy participants could report that the CS predicted that the US was coming ([Figure 10.12B](#)). The patient with amygdala damage could also report the details of the conditioning experiment quite well; specifically, she knew that the CS predicted the US—even though she had not generated any SCRs to the CS (as you saw in [Figure 10.10B](#)). In contrast, the patient with hippocampal damage, who had learned to produce the SCR, could not report any details of the conditioning experiment. This is consistent with the general expectation that a hippocampal lesion does not prevent simple classical conditioning but abolishes the ability to form new episodic memories about the context in which that conditioning occurred.

More recently, researchers have used optogenetics to show a direct influence of hippocampal contextual learning in healthy animals. In one study, rats were first given aversive shocks in a conditioning chamber and were then placed in a new context, where they had never experienced shock. There they behaved normally, curiously exploring their new environment. Next, with the rats still in the new environment, the researchers stimulated the same hippocampal neurons that had been active in the conditioning context—and the rats froze ([Liu et al., 2012](#)). In effect, the optogenetic stimulation reactivated the hippocampal memory for the conditioning context—producing a fear response (freezing) in the rats, just as if they had been returned to the conditioning context.

This ability of hippocampal contextual memory to influence emotional responding is part of the reason why returning to the location (or context) where an emotional experience occurred is often enough to evoke an emotional response. For instance, when people return to a place where

they experienced intense sadness or fear, they are sometimes struck with a fresh wave of grief or terror. This wave can include all the components of emotion: conscious feelings as well as physiological responses (e.g., heart rate and skin conductance changes) and overt behaviors (e.g., freezing or crying). Similarly (and more happily), couples celebrating an anniversary sometimes choose the restaurant where they had their first date because returning to the context where they once experienced strong feelings of romance can help evoke those same emotional responses again.

Conscious Feelings and the Frontal Lobes

So far, you have read that the amygdala plays an important role in learning and generating conditioned emotional responses that can be assessed as physiological responses (e.g., changes in heart rate and skin conductance) and as overt behaviors (e.g., freezing in response to threat). But remember, there is a third component to emotions: conscious feelings.

It is a matter of intense debate among researchers whether the amygdala plays a role in conscious feelings of emotion ([Fanselow & Pennington, 2018](#); [LeDoux & Pine, 2016](#)); however, it is widely accepted that the frontal lobes play an important part. As described in [Chapter 9](#), the *frontal lobes* of the cortex are often considered the seat of executive function, such as planning and decision making. The frontal lobes—particularly the prefrontal cortex, which lies at the extreme front of the mammalian brain—are also very much involved in social behavior, and appropriate social behavior demands the ability both to express emotion and to recognize and respond to expressions of emotion in others ([Dixon, Thiruchselvam, Todd, & Christoff, 2017](#)).

Humans with damage to the frontal lobes often exhibit less frequent and less intense facial expressions ([Kolb & Taylor, 1981](#)). These individuals may show a general disruption of emotion and mood, which can manifest as social withdrawal and loss of normal emotional display. (The old medical procedure of prefrontal lobotomy was a way of making chronically agitated people “calmer” by destroying parts of the frontal lobes so as to reduce emotional expression.) Other individuals with frontal lesions experience the opposite extreme, heightened emotionality, and exhibit inappropriate social behavior (profanity, public masturbation, and so on) and rapid mood swings, including violent bouts of anger and aggression for no discernible reason. Apparently, the frontal lobes help us maintain a balance between too little emotion and too much.

The prefrontal cortex also plays a role in helping people “read” the expression of emotion in others. In one study, volunteers viewed photos of neutral or fearful human faces (such as the “fearful” face you saw back in [Figure 10.1](#)). Meanwhile, researchers measured skin conductance responses and observed brain activation with fMRI ([L. Williams et al., 2001](#)). By now, you should be completely unsurprised to hear that amygdala activation was greater while participants viewed the fearful faces than while they viewed the emotionally neutral faces. The novel finding was that fearful faces also caused more activity in the medial prefrontal cortex than did emotionally neutral faces. These data are consistent with the idea that this area of the prefrontal cortex is active during emotional processing, helping to interpret the meaning of emotional stimuli, such as other people’s facial displays of emotion.

Several researchers have further proposed that the medial prefrontal cortex allows us to process emotional stimuli in a manner appropriate to the context in which the stimuli occur ([Hornak et al., 2003](#); [Kolb & Taylor, 1990](#); [Rolls, 1999](#)). For example, your emotional reaction to seeing a large snake should be very different depending on whether you encounter the snake as you walk through the woods or through the zoo. When you spot the snake, your amygdala may trigger an emotional response, but your hippocampus provides information about the context (“zoo ... no danger here”), and based on this information your medial prefrontal cortex will dampen the emotional response. In effect, the medial prefrontal cortex tells the amygdala not to bother because the snake *in this particular context* is no threat. Consistent with this idea, experiments show that electrically stimulating the medial prefrontal cortex in rats “turns off,” or inhibits, the normal emotional response provoked by amygdala stimulation ([al Maskati & Zbrozyna, 1989](#); [Quirk, Likhtik, Pelletier, & Paré, 2003](#)).

Remember the finding described at the start of this chapter that Japanese students tended to mask their emotional reactions in the presence of an authority figure? Most likely, the students were successfully using their frontal cortex to inhibit facial expressions in a context where it was considered polite not to show discomfort.

Emotional Memory in the Brain

We have seen that emotions have a powerful effect on how we store and retrieve episodic and semantic memories. Research shows that the amygdala, which is so important to emotional learning and responding, contributes to these processes as well.

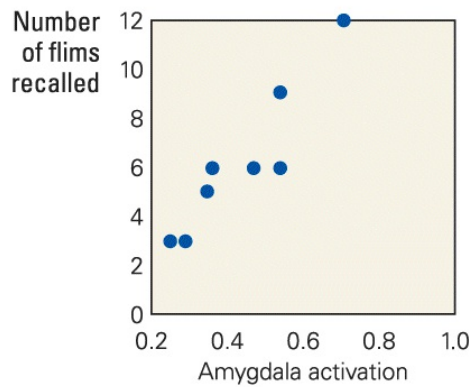
Amygdala Activation Can Strengthen Memory Encoding

Remember the experiment in which a slide show accompanied narration of either an emotionally arousing story or a neutral story? As you saw in [Figure 10.6](#), healthy people recalled more details of the emotionally arousing story, particularly from the dramatic middle part, than of the neutral story. By contrast, a patient with amygdala damage recalled the beginning and end of the story about as well as did healthy controls but showed no physiological arousal during the emotional middle section and no tendency, later, to recall that material better ([Cahill et al., 1995](#)). It was not that the patient couldn't remember the story but rather that the memory of the middle part didn't get an emotional "boost." This finding implies that the amygdala may provide a signal to strengthen the storage of information in declarative memory.

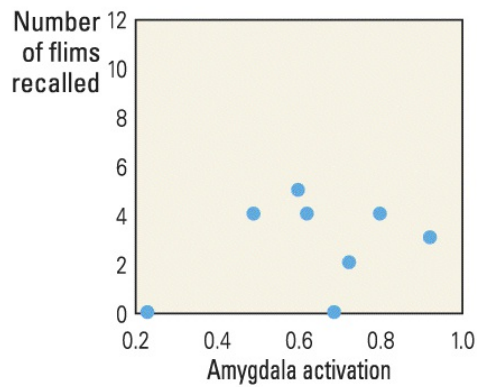
Even in healthy brains, the degree of amygdala activation may reflect how effectively information is processed and stored in memory. In an early demonstration of this idea ([Cahill et al., 1996](#)), healthy volunteers were shown 12 brief film clips of highly emotional events (e.g., violent crimes, animal mutilation) and 12 brief film clips of emotionally neutral events (e.g., courtroom proceedings, travelogues). Meanwhile, researchers used functional neuroimaging to record the volunteers' brain activity. Three weeks later, participants were given a surprise test, in which they were asked to recall as many film clips as possible.

As you would expect, participants had better memory for the emotional films than for the neutral films (recalling, on average, about 6 or 7 emotional films but only 2 or 3 neutral films). In addition, individuals who had high levels of amygdala activation while viewing the emotional film clips tended to recall more of those clips than did individuals with weaker amygdala activation ([Figure 10.13A](#)). There was no significant association between degree of amygdala activation and memory for the neutral film clips ([Figure 10.13B](#)). [Chapter 7](#) introduces the *subsequent memory* paradigm, in which participants with more hippocampal activation during memory encoding subsequently have better memory for the studied material. The effect summarized in [Figure 10.13](#) is similar: more amygdala activation during encoding is associated with better memory later—but, in the case of the amygdala, this benefit applies only for material with emotional content.

A Emotional film clips



B Neutral film clips



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FIGURE 10.13 Amygdala activation and memory for emotional material Healthy participants saw short emotionally arousing films while their brain activity was recorded using PET. (A) Later, the participants who had shown the highest amygdala activity during viewing remembered more details about the emotionally arousing films. (B) There was no significant relationship between amygdala activity and memory for films containing emotionally neutral material.

Research from [Cahill et al., 1996](#).

Description

The readings on the vertical axis range from 0 to 12 in increments of 2 and the readings on the horizontal axis range from 0.2 to 0.8 in increments of 0.1. The data in the two graphs is as follows.

A. Emotional film clips: the plots correspond to the following points: (0.25, 3), (0.3, 3), (0.35, 5), (0.4, 6), (0.5, 6), (0.6, 5.9), and (0.7, 12).

B. Neutral film clips: the plots correspond to the following points: (0.2, 0), (0.4, 4), (0.5, 5), (0.51, 4), (0.57, 0), (0.6, 2), (0.7, 4), and (0.75, 3).

All the above data are approximate.

Other studies have documented that the amygdala is also activated while participants view pleasant pictures and that greater amygdala activity during encoding of specific pictures is correlated with better recognition of those specific pictures later ([Canli, Zhao, Brewer, Gabrieli, & Cahill, 2000](#); [Hamann, Ely, Grafton, & Kilts, 1999](#)).

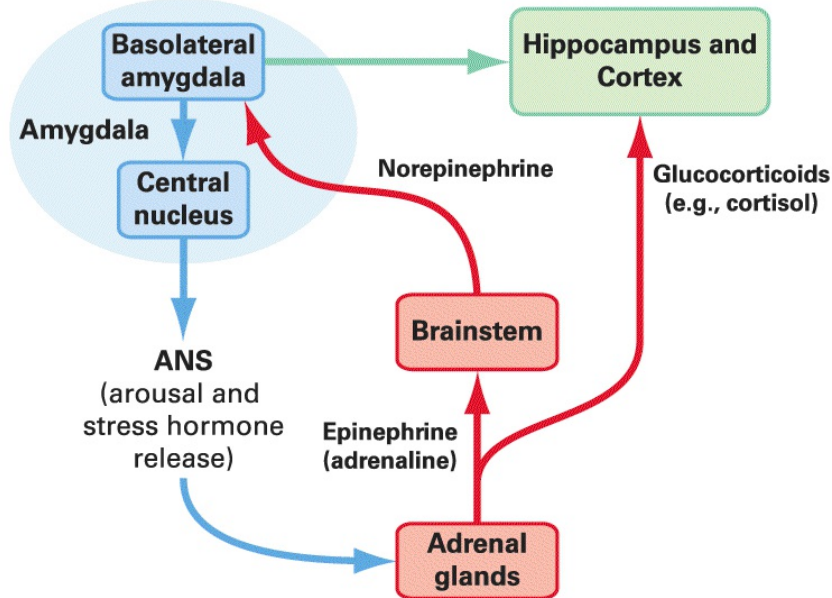
The degree of amygdala activity during encoding may also help determine whether new information gets stored as an episodic memory or as a semantic memory. [Chapter 7](#) defines *episodic memory* as information we “remember” and *semantic memory* as information we “know.” Episodic memory has supporting contextual information: we remember the time and

place at which we experienced the event that produced the memory. To probe the difference between episodic and semantic memory, healthy adults are shown a series of emotional photographs and are then, at a later time, asked which ones they recognize. For the recognized pictures, the participants are also asked whether they “remember” having previously studied that picture or merely “know” that the picture looks familiar (with no specific memory of having studied it during an earlier session).

Functional neuroimaging has been used to show that amygdala activity is higher both at encoding ([Dolcos, LaBar, & Cabeza, 2004](#)) and at recognition ([Sharot, Delgado, & Phelps, 2004](#)) of emotionally arousing photographs that are judged as “remembered” than of pictures that are judged as merely “known.” One interpretation of these findings is that emotional arousal (and amygdala activation) promotes encoding of contextual details in the hippocampus, creating a subjective sense of “remembering” and causing the information to be stored as an episodic rather than semantic memory ([LaBar, 2007](#)). This in turn could be why we often have such a vivid sense of recollection for highly emotional events: flashback memories may form when episodic memories of highly emotional events are tagged with a particularly strong feeling of “remembering.”

Stress Hormones Can Strengthen Memory Encoding

Given the associations between amygdala activation and strong memory, the next question is: Exactly how might the amygdala influence memory storage? As you saw in [Figure 10.9](#), one set of outputs from the central nucleus of the amygdala travels down to the ANS. [Figure 10.14](#) shows that pathway in more detail ([Garcia, 2017](#); [McGaugh, 2013, 2015](#)). The ANS signals the adrenal glands (which are located above the kidneys) to release stress hormones, including glucocorticoids and epinephrine, into the bloodstream. Epinephrine helps mediate the various components of the fight-or-flight response, including increased heart rate and dilation of blood vessels, to facilitate blood flow to the brain and muscles.



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FIGURE 10.14 Some pathways by which the amygdala can modulate memory storage in the hippocampus Emotional arousal signaled by outputs from the central nucleus of the amygdala to the autonomic nervous system (ANS) causes the adrenal glands to release stress hormones such as glucocorticoids and epinephrine. Glucocorticoids can enter the brain and affect many brain structures, including the hippocampus. Epinephrine cannot easily enter the brain. Instead, epinephrine activates brainstem nuclei that project norepinephrine to many brain structures, including the basolateral nucleus of the amygdala, which in turn projects to the hippocampus.

Based on information from [McGaugh, 2003](#).

Description

The Basolateral amygdala and the central nucleus of Amygdala lead to A N S (arousal and stress hormone release). A N S leads to the adrenal gland which releases epinephrine (adrenaline) into the brain stem. Brainstem projects norepinephrine into basolateral amygdala, which leads to the hippocampus and cortex. The adrenal glands also release glucocorticoids (example: cortisol) which projects to hippocampus and cortex. The Basolateral amygdala and the central nucleus of the Amygdala are marked within a shaded circle.

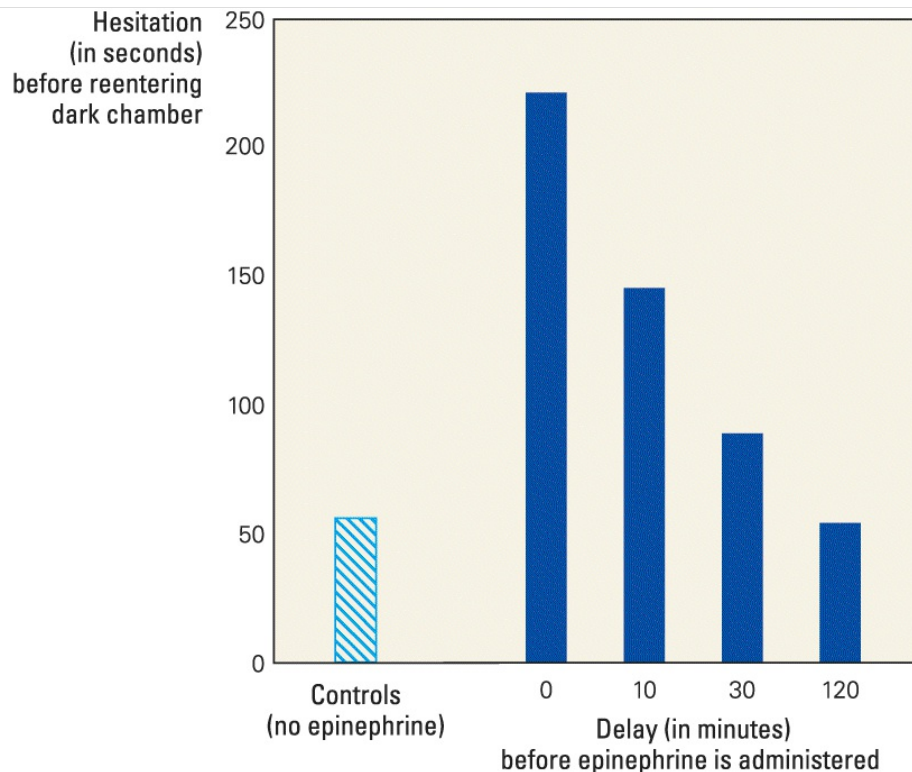
But epinephrine cannot affect the brain directly. The brain has a defense called the *blood–brain barrier*, a membrane that controls passage of substances from the blood into the central nervous system, including the brain. This protects the brain from many chemicals that might otherwise enter and harm it. Epinephrine cannot easily cross the blood–brain barrier, but it can activate brainstem areas that produce the chemically related neurotransmitter [norepinephrine](#), which can cross the blood–brain barrier.

norepinephrine

A neurotransmitter that is chemically related to epinephrine but that (unlike epinephrine) can cross the blood–brain barrier and affect brain function.

In the brain, norepinephrine projects to many brain areas, including the basolateral amygdala. You can think of this pathway as “reporting back” to the amygdala that the body is experiencing arousal. The basolateral amygdala, in turn, contains neurons that project out of the amygdala to the cortex and hippocampus. Norepinephrine tends to activate neurons of the basolateral amygdala in rhythmic waves ([Paré, 2003](#)), and this rhythmic activity may flow outward to promote similarly rhythmic activation in large groups of cortical and hippocampal neurons, leading to LTP between the coactive neurons (again, “neurons that fire together wire together”). In this way, norepinephrine may indirectly promote strong learning in the cortex and hippocampus.

Consistent with this idea, numerous studies have shown that healthy participants given injections of epinephrine during or immediately after learning will often exhibit better memory for the studied material (for review, see [Shields, Sazma, McCullough, & Yonelinas, 2017](#)). Similarly, norepinephrine administered immediately after training will typically enhance memory in nonhuman animals (for review, see [McGaugh, 2015](#)). For example, [Figure 10.15](#) shows that control rats, after having received a shock in a dark chamber, will hesitate about 60 seconds before daring to enter the dark chamber again (pale blue striped bar); it also shows what happens if the rats are given injections of epinephrine after the foot shock (dark blue bars). If the epinephrine is administered immediately (0 seconds) after the foot shock, rats hesitate for more than 200 seconds before reentering the dark chamber. As outlined in [Figure 10.14](#), epinephrine triggers norepinephrine release to the basolateral amygdala, which in turn stimulates learning in the cortex and hippocampus—making the rats remember more strongly that the dark chamber is a dangerous place. The effects of epinephrine are greatest if injection occurs immediately after the foot shock, but epinephrine can still boost memory if administered 10 or 30 minutes after the training session ([Gold & van Buskirk, 1975](#)). By 120 minutes after the foot shock, the time window for manipulating the memory seems to have closed, and epinephrine injections have no effect.



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FIGURE 10.15 Stress hormones can strengthen memory If control rats are given a shock the first time they enter a dark chamber, they will delay an average of about a minute before reentering that chamber (pale blue bar). Rats given an injection of epinephrine immediately (0 minutes) after the training session will delay much longer, indicating that the post-training epinephrine increased their memory for the episode. The effects are time sensitive; an injection given 2 hours (120 minutes) after training has little effect ([Gold & van Buskirk, 1975](#)).

Information from [McGaugh, 2003](#).

Description

The readings on the vertical axis range from 0 to 250 in increments of 50. The data in the graph reads as follows.

Controls (no epinephrine): 60 seconds, Delay before epinephrine is administered: 0 minutes-225 seconds of hesitation, 10 minutes-145 seconds of hesitation, 30 minutes- 80 seconds of hesitation, and 120 minutes-50 seconds of hesitation.

All the above data is approximate.

On the other hand, blocking or reducing norepinephrine can reduce memory for emotional material ([McGaugh, 2015](#)). Recall the study from [Figure 10.6](#), in which healthy participants saw a slide show and had better memory for the emotional middle part of the story than for the emotionally neutral beginning and end. In one variant of the experiment, before the slide show was presented, some participants were given the drug propranolol, which blocks norepinephrine,

while others were given an inactive placebo ([Cahill, Prins, Weber, & McGaugh, 1994](#)). Later, the participants who had received the placebo showed the normal pattern of remembering the emotionally charged middle part best (just like the participants in [Figure 10.6](#)). In contrast, the participants who had been given propranolol remembered the middle part no better (and no worse) than the emotionally neutral beginning and end parts. These results also help explain why patients with amygdala damage fail to show enhanced memory for emotional information. Without norepinephrine to strengthen memory encoding for the emotionally arousing material, that material is stored neither more nor less strongly than any other information.

[Figure 10.14](#) shows that, under ANS activation, the adrenal glands also release glucocorticoids. Unlike epinephrine, these stress hormones can enter the brain directly. The hippocampus has a particularly high concentration of glucocorticoid receptors ([McEwen & Sapolsky, 1995](#)), and low levels of glucocorticoids are able to facilitate LTP and encourage dendritic growth in the hippocampus, improving memory formation and recall ([McEwen, 1999](#)). Studies have shown that low levels of glucocorticoids (e.g., cortisol in humans) administered immediately after encoding can improve memory for emotional material ([Buchanan & Lovallo, 2001](#); [Cahill & Alkire, 2003](#)).

Unfortunately, *high* levels of glucocorticoids often *impair* memory encoding ([de Quervain, Roozendaal, & McGaugh, 1998](#); [de Quervain, Roozendaal, Nitsch, McGaugh, & Hock, 2000](#)). This may be because high levels of glucocorticoids can overexcite the hippocampus, thereby interfering with learning ([Benjamin, McKeachie, Lin, & Holinger, 1981](#)). Long-term exposure to high levels of glucocorticoids, as occurs in individuals under extreme and chronic stress, may even cause neuronal death in the hippocampus ([McEwen, 1997](#); [Sapolsky, 1996](#)). (For ways to identify and control sources of stress in your daily life, see “[Learning and Memory in Everyday Life](#)”.)

LEARNING AND MEMORY IN EVERYDAY LIFE

How Much Stress Is Too Much?

As you have read in this chapter, a little stress can be good for memory, helping us identify and strongly encode information that is important for success and survival. But a high level of stress, particularly if it persists for a long time, can impair memory encoding and even cause damage to hippocampal neurons ([Bremner, 1999](#)).

How much stress is too much? When do memory-promoting “low” levels of arousal transition to memory-impairing “high” levels? Unfortunately, there is no easy answer: each individual seems to have a different breaking point ([Kemeny, 2003](#)).

Some individuals, such as stockbrokers and emergency room nurses, seem to thrive on excitement and challenge, whereas others fall to pieces at the slightest setback.

Furthermore, each individual's response to stress depends partly on whether he or she feels helpless to control it. You may have felt the difference yourself. On a day when you feel excited and in control, the feelings of arousal may rev up your brain and help you perform your best. But on days when you feel overwhelmed and “stressed out,” you may notice yourself making more memory slips than usual. During periods of chronic stress, such as a serious illness or a relationship problem, the constant flood of stress hormones through your body can affect not only your memory but your energy, digestion, and immune system, making you more vulnerable than usual to infection and disease ([Sapolsky, 1996](#)).

If you are constantly feeling stressed, start by making a list of the things that bother you and then look for ways to change (control) some of them. Even the act of making the list can help you reassert a sense of control over your life.

You can also take up specific practices that reduce stress, such as meditation, yoga, prayer, or exercise. These activities provide a temporary escape from the ups and downs of daily life. Performed on a regular basis, they may have medical benefits—and may even help get your hippocampus back to normal ([Davidson et al., 2003](#)).

Stress Hormones Can Impair Recall

In addition to affecting memory encoding, stress hormones can affect retrieval. But whereas modest doses of stress hormones at the time of learning will often improve new memory formation (as you saw in [Figure 10.15](#)), the presence of stress hormones at recall will often impair the retrieval of older memories.

For example, humans or rats given glucocorticoids are often impaired at recalling previously learned information (for review, see [de Quervain, Schwabe, & Roozendaal, 2017](#)). This may be because under normal conditions, the presence of high levels of stress hormones signals that something important is going on and that the brain should shift to learning about whatever is causing the stress rather than recalling old information ([Meir Drexler & Wolf, 2017](#)).

If you have ever studied hard for an exam but then “blanked out” during the test itself, glucocorticoids may be to blame. Even if you successfully encoded the material, high levels of stress hormones may have interfered with your ability to retrieve that information. Later, after the exam, as your stress levels returned to normal levels, your hippocampus resumed normal function—and you may have remembered all those answers that eluded you during the test.

Stress Hormones Can Either Strengthen or Impair

Reconsolidation

As discussed in [Chapter 7](#), memories are not formed instantaneously but remain malleable throughout a *consolidation period*, during which time they are vulnerable to such interventions as electroconvulsive shock or head injury. [Chapter 7](#) also describes *reconsolidation*, in which reactivation or retrieval of an old memory can make it vulnerable all over again. In the case of an emotional memory, the mere act of recollection can trigger a fresh dose of stress hormones that could strengthen (or weaken) the neural circuits encoding that memory, possibly making emotional memories even more susceptible than other types of memory to changes during reconsolidation. This suggests one explanation for the creep of small distortions into flashbulb memories: the memory might initially be accurate, but each time it is recalled, tiny details might be altered, and over time, the memory could become quite different from its original form.

Emotion researcher James McGaugh has suggested that there is an important reason why emotional systems in animals might have evolved the ability to modulate memory after the fact. In many cases, the importance of a particular event might not be immediately apparent. For instance, if a pigeon drinks colored water that contains a nausea-inducing agent, the symptoms of sickness may not appear for some time—but once those symptoms do appear, it is very important for the sick pigeon to be able to encode a strong memory of the colored water. Similarly, a child who scribbles on the wall may not be punished until hours later, when her parents find out; in this case, the child must be able to reach back in time and strongly associate the earlier action with the later consequence. In such cases, the ability of stress hormones to affect previously acquired memories allows the amygdala to tinker with the strength of the memory later, when the delayed consequences become apparent ([McGaugh, 2003](#)).

TEST YOUR KNOWLEDGE

Brain Substrates of Emotion

Emotional responses depend on a complex interplay between many brain areas, including the amygdala, hippocampus, and prefrontal cortex. Read the story below and identify some ways these brain areas could be contributing at each of the four steps identified by a number in parentheses. ([Answers appear at the end of the chapter.](#))

Jared is afraid of heights. However, on a visit to Seattle, his friends say he should go to the top of the Space Needle for a stunning view of the city. On the elevator up, Jared feels the beginnings of a nervous reaction: his heart beats faster, his mouth feels dry, and his stomach tightens (1). He remembers when he was similarly terrified standing on the high diving board at school (2).

When he arrives at the outdoor observation area, Jared's first impulse is to flatten himself against the wall and stay as

far away from the edge as possible. But having come this far, he takes a few deep breaths, walks up to the glass barrier, and looks out over the city (3). In spite of everything, he is able to appreciate the beautiful view (4).

Interim Summary

- Emotion depends on many brain areas, and each brain area may contribute to more than one emotion.
- Part of the body's response to strong emotion comes from activation of the amygdala; outputs from the central nucleus of the amygdala drive many of the behavioral and physiological components of emotional responses. Animals (including people) with amygdala damage show reduced emotionality and a reduced ability to learn conditioned emotional responses.
- The basolateral amygdala plays a role in learning conditioned emotional responses. The hippocampal region can influence emotional learning by providing information about the context in which the learning occurs.
- The frontal lobes play a role in humans' ability to display emotions appropriately and to read emotional expressions in others, as well as in the ability to process emotional stimuli in a manner appropriate to the context in which they are experienced.
- The basolateral amygdala also modulates storage of emotional memories, through its projections to the cortex and the hippocampal region. Memories can be formed in the cortex and hippocampus without amygdala input, but if the emotional system is triggered, then memory formation will be stronger.
- Stress hormones (e.g., epinephrine, norepinephrine, glucocorticoids) typically strengthen memory encoding but impair memory recall. They can either strengthen or impair memories during reconsolidation.

10.3 Clinical Perspectives

Much of the research covered in this chapter has focused on negative emotions—particularly fear—in part because negative emotions have a tremendous impact on our health and well-being. Long-term unremitting fear and anger, leading to chronic activation of the ANS, can produce physiological problems such as high blood pressure and immune system suppression. Negative emotions can also cause psychological problems. Two of these in particular—phobias and posttraumatic stress disorder—may involve learning and memory processes in their development and in their successful treatment.

Phobias

A **phobia** is an excessive and irrational fear of an object, a place, or a situation that leads to anxiety and panic attacks. Currently, most phobias are classified into two categories: *specific phobias* and *agoraphobia*.

phobia

An excessive and irrational fear of an object, a place, or a situation.

Specific phobias are fears of particular objects or social situations. Examples include fear of closed spaces (claustrophobia), fear of heights (acrophobia), fear of snakes (ophidiophobia), and fear of spiders (arachnophobia). In many (but not all) cases, the phobia centers on an object or a situation in which a fear reaction might be justified. For example, it is appropriate to be alarmed by the sight of a snake since snake venom can be deadly. Similarly, a healthy fear of heights may keep you from falling off cliffs. But when a fear has reached a point where it interferes with daily life, it is classified as a phobia. For example, if a woman were so afraid of heights that she was unable to ride the elevator at work, or if a man were so afraid of snakes that he avoided sitting outside in his own backyard, these fears would be classified as phobias.



VCG/Getty Images

Common specific phobias include fear of snakes, fear of spiders, fear of heights, and fear of closed spaces. What differentiates a phobia from a normal, healthy response to danger?

Description

The bridge is constructed adjoining a rocky mountain, and the person seeks support from the rocky sides of the mountain. Underneath is a misty valley.

Whereas specific phobias center on particular objects or situations, *agoraphobia* involves a generalized fear of leaving home or familiar “safe” areas, usually because of fear of having a panic attack in public. During a panic attack, epinephrine is released in large amounts, triggering the fight-or-flight response, with symptoms that include rapid heartbeat, trembling, nausea, and dizziness. Fear of experiencing a panic attack, and of the social embarrassment associated with having a panic attack in public, can lead individuals with agoraphobia to go to great lengths to avoid open, public places. In the most severe cases, individuals with agoraphobia become completely housebound, some unable to leave home for years on end.

What Causes Phobias?

One theory about the formation of phobias is that they arise through classical conditioning.

[Chapter 1](#) introduces John Watson, the “father” of *behaviorism*, a school of psychology that focused on the study of observable behaviors. In one famous series of studies, Watson and his research assistant Rosalie Rayner studied fear conditioning in an 11-month-old boy known as Little Albert ([Watson & Rayner, 1920/2000](#)). Two months before the experiment began, Watson and Rayner had presented Albert with objects including a white rat, a rabbit, a bearded Santa Claus mask, and a burning newspaper. Albert had shown no fear responses to any of these objects, although he did react with apparent fear to a loud clanging noise.

To study fear conditioning, Watson and Rayner placed a white rat near Albert and allowed him to reach out toward it. As he did, they startled him with the loud clanging noise, and he began to cry. After several pairings of the rat and the noise, the researchers presented Albert with the rat and no noise; Albert now cried at the sight of the rat and tried to crawl away. The researchers concluded that Little Albert had been classically conditioned to associate the rat (a CS) with a fear-provoking loud noise (the US). This learning appeared to generalize to similar stimuli: Albert now reacted with fear to other white, furry objects, such as the rabbit and the Santa Claus mask, but not to other unrelated objects such as wooden blocks.



Benjmain Harris

While Little Albert was playing with a pet rat, John Watson and Rosalie Rayner paired the rat with a loud, frightening noise.

Albert developed a conditioned fear response to the rat. Watson and Rayner (visible in the photo) claimed that they had induced a phobia in Albert and that such classical conditioning could underlie the formation of real-world phobias. Assuming that Albert had indeed developed a phobia based on classical conditioning, how might Watson and Rayner have attempted to “cure” Little Albert before sending him home?

Shortly after, Little Albert and his mother moved away. There is no record of how severe Albert’s fear of rats was, how long it persisted, or whether it generalized to rats encountered outside of Watson’s lab ([Beck, Levinson, & Irons, 2009](#); [Harris, 1979](#)). Today, of course, ethical guidelines would preclude experiments like the one conducted on Little Albert. At a bare minimum, Watson and Rayner should have made an effort to extinguish Albert’s conditioned fear before he left the study. Over the years, scientists have continued to debate Little Albert’s true identity and the ethics of his treatment in the hands of Watson and Rayner ([Beck et al., 2009](#); [Harris, 1979](#); [Powell, Digdon, Harris, & Smithson, 2014](#)), but the experiment did raise some interesting questions about classical conditioning and phobias. Maybe some naturally arising phobias also reflect classical conditioning; perhaps some people develop claustrophobia after a traumatic experience such as being locked in a closet and others develop a fear of dogs after being attacked by an out-of-control pet.

Not everyone who has a fear-evoking experience develops a phobia. One study reported that two-thirds of people with dog-related phobias can recall dog-related trauma (e.g., being bitten), but so can two-thirds of people with no fear of dogs ([DiNardo, Guzy, & Bak, 1988](#)). Nor does everyone with a phobia report an initial fear-evoking event: some people have a terror of snakes despite never having come into contact with a living snake, and others are morbidly afraid of flying without ever having been in an airplane.

One possible explanation is that some phobias may be conditioned through social transmission, which is discussed further in [Chapter 11](#). For example, a person can develop a fear of flying after seeing televised reports of an airplane crash or a fear of snakes after seeing others react with fear at mention of a snake. Monkeys show socially transmitted fear, too. A monkey raised in the lab will not react with fear the first time it sees a snake. But if the lab-reared monkey is caged next to another monkey that it observes reacting with fear at the sight of a snake, the lab-reared monkey will begin to show the same fear response ([Mineka & Cook, 1988](#)). Possibly, the sight of a frightened neighbor is itself a fear-evoking US, and the object that evokes that fear becomes a CS in the observer monkey.

Therapy for Phobias

If some phobias arise through conditioning, can they be extinguished in the same way as ordinary conditioned responses—that is, by repeated exposure to the CS with no US? In many cases, they can. In [systematic desensitization](#) therapy for phobias, successive approximations of the CS are presented while the patient learns to remain relaxed; eventually, even the CS itself does not elicit a fear response ([Kazdin & Wilcoxon, 1976](#); [Linden, 1981](#)). For example, a person who is afraid of snakes may first be presented with some snake-shaped item (e.g., a hose, rope) different enough from a real snake that no fear reaction is evoked. In successive sessions, the patient is progressively exposed to, say, a toy rubber snake, then a photograph of a real snake, then a person holding a live snake. These steps are taken so gradually that they do not evoke a fear reaction. Eventually, the patient may be asked to touch a live snake, then to hold it himself. As you might imagine, systematic desensitization therapy can be a slow and painstaking process, but it is often successful and long-lasting.

systematic desensitization

Therapy for phobias in which successive approximations of the fear-evoking stimulus are presented while the patient learns to remain relaxed; eventually, even presentation of the stimulus itself does not elicit a fear reaction.

Sometimes, however, it is dangerous, inconvenient, or very expensive to expose the patient (and the therapist!) to the object of the phobia. For instance, if the patient is afraid of flying, systematic desensitization might require her to practice approaching an airport, then practice boarding and sitting on a grounded plane, then ride a plane as it taxis along the runway, and finally work up to an actual flight. This would be prohibitively expensive for most people. Instead, the therapist may recommend virtual-reality therapy using computer-generated virtual environments. The patient can “experience” sitting in an airplane seat, gradually getting used to the sights and sounds of an aircraft and even taking simulated flights, before spending the energy and money to board a real plane. Virtual-reality therapy, particularly in combination with relaxation training and cognitive therapy, can be very effective in treating phobias ([Emmelkamp et al., 2002](#); [Muhlberger, Herrmann, Wiedeman, Ellgring, & Pauli, 2001](#)).

As you might expect, some studies have reported hyperactive amygdala in individuals with phobias; there have also been reports of abnormalities in the anterior cingulate cortex and insula—which, as explained in [Chapter 5](#), are important for helping us determine what is aversive and what we are motivated to avoid. It is not too surprising that these brain areas might be overactive

in individuals with phobias. Interestingly, several studies have now reported that, when patients undergo therapy to treat their phobias, these same fear-related brain structures show reduced activity ([Galvao-de Almeida et al., 2013](#); [Lipka, Hoffmann, Miltner, & Straube, 2014](#)). These findings suggest that successful treatment of phobias helps patients' brains recalibrate, so that the previously feared object or situation is no longer interpreted as being quite so threatening.

TEST YOUR KNOWLEDGE

Classical Conditioning and Phobias

At least some phobias appear to arise through classical conditioning, when a previously neutral stimulus is paired with a strongly aversive or fear-evoking event. Read the scenario below and see if you can identify the CS, US, and CR. ([Answers appear at the end of the chapter.](#))

Nancy has recently lost her job and is worried that she will not be able to provide for her children. This worry is exacerbated when she goes to the grocery store and sees the high prices. While at the store, Nancy has a panic attack, a sudden and overwhelming feeling of intense fear. She feels like the room is spinning; her heart pounds and she can't seem to get enough air. After a few minutes, the episode passes, but she is deeply shaken by the experience. In the days that follow, she is terrified of having another panic attack, especially in public. She finds herself dreading the thought of returning to the grocery store and reassigns the household chores so that her teenaged children take care of the grocery shopping.

Posttraumatic Stress Disorder

Following the 9/11 terrorist attacks, thousands of counselors traveled to New York City to offer their services to survivors, families, and rescue workers. In addition to these volunteers, \$23 million in federal funds was allocated to Project Liberty, a program that provided free counseling to New Yorkers.

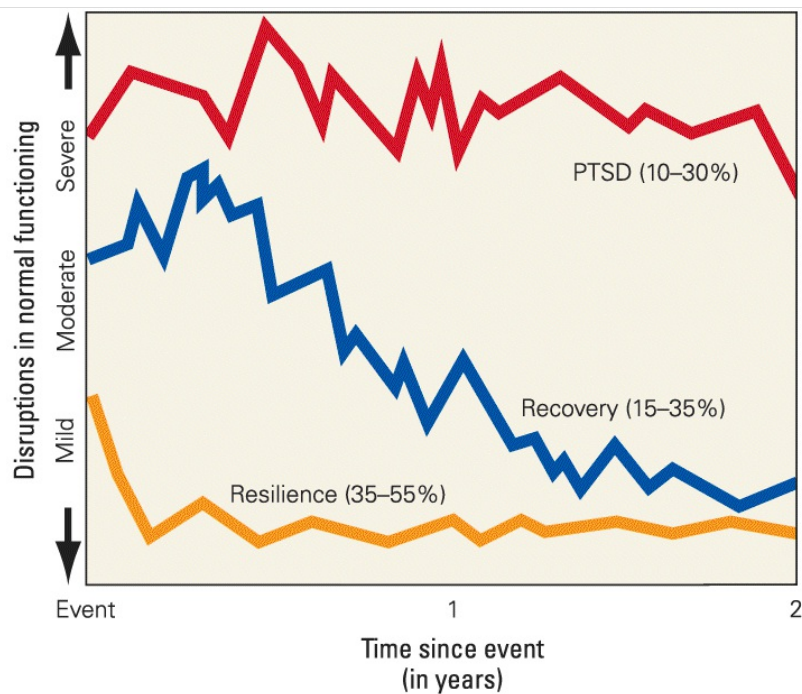
The rationale for this convergence of mental health professionals and funding was the expectation that in the wake of the attacks, many New Yorkers would develop **posttraumatic stress disorder (PTSD)**. PTSD is a psychological disorder that can develop after a traumatic event (e.g., combat, rape, natural disaster); the symptoms include re-experiencing the event (through intrusive recollections, flashbacks, or nightmares), avoidance of reminders of the trauma, emotional numbing, and heightened anxiety ([Shalev, Liberzon, & Marmar, 2017](#)).

posttraumatic stress disorder (PTSD)

A psychological syndrome that can develop after exposure to a traumatic event (e.g., combat, rape, natural disaster); symptoms

include re-experiencing the event (through intrusive recollections, flashbacks, or nightmares), avoidance of reminders of the trauma, emotional numbing, and heightened anxiety.

Such fear reactions are a perfectly normal response to distressing events; but for most people exposed to trauma, the fear reactions subside with time, a phenomenon termed “resilience” ([Figure 10.16](#)). For individuals with PTSD, the fear reactions persist for months or years—sometimes even worsening over time. One report estimated that, of 3,271 workers who evacuated the World Trade Center towers during the 9/11 attacks, about 15% had PTSD 2 to 3 years later ([Perlman et al., 2011](#)). Another report, which examined data from almost 5 million U.S. veterans who had served during the conflicts in Iraq and Afghanistan, estimated that about 23% of these veterans had PTSD ([Fulton et al., 2015](#)).



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FIGURE 10.16 Patterns of psychological recovery after a traumatic event Immediately after experiencing a traumatic event (e.g., rape, combat exposure, a terrorist attack), most people show a disruption in normal functioning due to fear reactions; these may include heightened anxiety, nightmares, flashbacks, and avoidance of reminders of the trauma. In many people, these fear reactions dissipate with time (a pattern described as “resilience”), although some show more severe fear reactions that may take months or years to subside (“recovery”). But some individuals develop posttraumatic stress disorder (PTSD), with symptoms that persist for months or years after the event.

Hypothetical data; information from [Bonanno, 2005](#).

Description

The events on the vertical axis are labeled as mild, moderate, and severe starting from the bottom to top. The time since the event on the horizontal axis reads 1 and 2. The graph shows three curves representing resilience (35 to 55 percent), recovery (15 to 35 percent), and P T S D (10 to 30 percent). All three curves have intermittent peaks and falls. The resilience curve corresponds to a mild event, the recovery curve corresponds to a moderate event, and the P T S D curve corresponds to a severe event.

Predicting and Preventing PTSD

Many neurobiological mechanisms appear to be disrupted in patients with PTSD. These include an overactive amygdala, underactive hippocampus and prefrontal cortex, and disruptions in the level and timing of stress hormone release ([Liberzon & Sripada, 2008](#)). All of these factors are likely to play roles in the development and expression of PTSD symptoms.

One prominent theory of PTSD assigns a central role to classical conditioning ([Liberzon & Sripada, 2008](#); [van der Kolk, 1994](#)). By definition, patients with PTSD have experienced an event that causes a strong fear response, such as feelings of helplessness or terror. This event may function as a US, and it may be such a strong and effective US that any other co-occurring stimuli become strongly associated with it. Thus, sights, sounds, and smells may all become conditioned stimuli. An individual with a hyperactive amygdala may learn the CS–US associations particularly strongly. Later, when any of these CSs is experienced again, it may evoke the memory of the US—and trigger a conditioned fear response.

This fear reaction is not abnormal in the aftermath of a traumatic event, but for most people, re-experiencing these CSs without recurrence of the traumatic US should gradually lead to extinction of the fear response in the weeks and months after the trauma. These resilient individuals do not forget their trauma, nor the negative emotions associated with that trauma, but the CSs are no longer able to evoke the fear CR. In contrast, individuals with PTSD may fail to extinguish the normal fear response to stimuli associated with the traumatic event ([Rothbaum & Davis, 2003](#)). In fact, in the laboratory, individuals with PTSD do typically show faster learning but slower extinction of conditioned emotional responses ([Orr et al., 2000](#); [Peri, Ben-Shakhar, Orr, & Shalev, 2000](#)).

[Chapter 7](#) introduces the phenomenon of *directed forgetting*: people who are explicitly instructed to forget information do indeed show worse recall of that information later. Functional imaging

(fMRI) has shown that the prefrontal cortex plays a key role in controlling directed forgetting by suppressing the hippocampus, which in turn impairs retention of the information. Perhaps PTSD reflects a reduced ability of the prefrontal cortex to inhibit hippocampal function and control the recall of unwanted memories ([Anderson & Levy, 2009](#)). In fact, in the lab, individuals with PTSD who are given a directed forgetting task that involves emotional pictures are indeed less successful at intentionally forgetting the images compared to participants with no PTSD ([Catarino, Küpper, Werner-Seidler, Dalgleish, & Anderson, 2015](#)). This could be why patients with PTSD experience intrusive memories, nightmares, and flashbacks: a wide variety of stimuli start to trigger recall, and the patients' prefrontal cortex cannot stop the traumatic memories in their tracks.

Therapy for PTSD

Consistent with the theory that PTSD involves a failure of extinction, some of the most widely used treatments for PTSD include exposing the patient to cues that trigger his anxiety—but doing so in the absence of actual danger. The purpose is to encourage extinction of the abnormally strong fear response ([Rothbaum & Davis, 2003](#)). Such *extinction therapy* for PTSD may require the patient to repeatedly imagine and describe the feared situations under the guidance of a therapist. Virtual-reality techniques also prove useful—for example, allowing veterans to navigate through a simulated version of the region where they had their most stressful combat-related experiences, thus undergoing repeated exposure to the fear-evoking stimuli within the safe context of a therapist's office. Under these conditions, some individuals experience gradual reduction of PTSD symptoms.



John J. Kruzel/615 collection/Alamy Stock Photo

Several months after returning from combat duty in Iraq and Afghanistan, up to 20% of U.S. soldiers met the diagnostic criteria for PTSD ([Hoge et al., 2004](#)). Some soldiers experiencing PTSD are helped by therapy designed to encourage extinction of fear responses, such as virtual-reality (VR) therapy providing the sights and sounds of a “virtual Iraq” or other war zone environment, without the actual danger. If you were designing a VR system to treat PTSD in a veteran who had experienced traumatic events during combat, what features might you try to include to make the extinction therapy as effective as possible? How would you assess its effectiveness?

Description

One of them has the visual reality equipment attached to his head, while the other soldier is displaying clips on a computer monitor next to him.

However, one limitation of extinction therapy for PTSD is that the extinction does not always generalize well ([Bouton, Westbrook, Corcoran, & Maren, 2006](#)). Rather, extinction may be limited to the context in which the extinction occurred. In the lab, if a rat learns a conditioned emotional response (e.g., freezing, blood pressure changes) to a CS that predicts a shock US, and then undergoes extinction training in which the CS no longer precedes the US, the conditioned response will gradually extinguish. But if the rat is later presented with the CS in a new context,

there is typically some return of the fear response—a phenomenon similar to the *spontaneous recovery* of defensive responding after habituation that is described in [Chapter 3](#). This recovery of fear responding occurs in humans also, but it is especially pronounced in participants with PTSD ([Milad et al., 2008](#); [Milad et al., 2009](#)). Outside the lab, a patient undergoing extinction therapy for PTSD may successfully reduce fear responses during the therapy session, but the fear responses and PTSD symptoms may rebound in other contexts, such as at home, at work, and in other everyday situations.

Some researchers have tried to use the principles of memory reconsolidation to treat PTSD. You read earlier that stress hormones can affect memory reconsolidation. Could they be used to help weaken traumatic memories? Several studies have attempted to test this idea by first having patients with PTSD reimagine traumatic events to reactivate the memories; the patients are then given drugs (e.g., propranolol, glucocorticoid antagonists) that have been shown to interfere with reconsolidation in rats. To date, these treatments appear to have had little or no effect on PTSD symptoms ([Argolo, Cavalcanti-Ribeiro, Netto, & Quarantini, 2015](#); [de Quervain et al., 2017](#); [Wood et al., 2015](#)), although it is possible that long-term treatment might be more effective.

Other research has suggested that extinction of fear responses can be improved by modifying the standard extinction procedures (for review, see [Dunsmoor, Niv, Daw, & Phelps, 2015](#)). For example, rather than try to reduce fear responses by pairing the CS with no US (as in standard extinction), it may be more effective to *replace* the US with a tone or other novel, non-threatening outcome ([Dunsmoor, Campese, Ceceli, LeDoux, & Phelps, 2015](#)). This new CS–tone learning may more effectively weaken the prior CS–US association (similar to the process of *retroactive interference* described in [Chapter 7](#)). It remains to be seen if these laboratory results can be applied in clinical settings to improve therapeutic outcomes for PTSD.

Predicting and Preventing PTSD

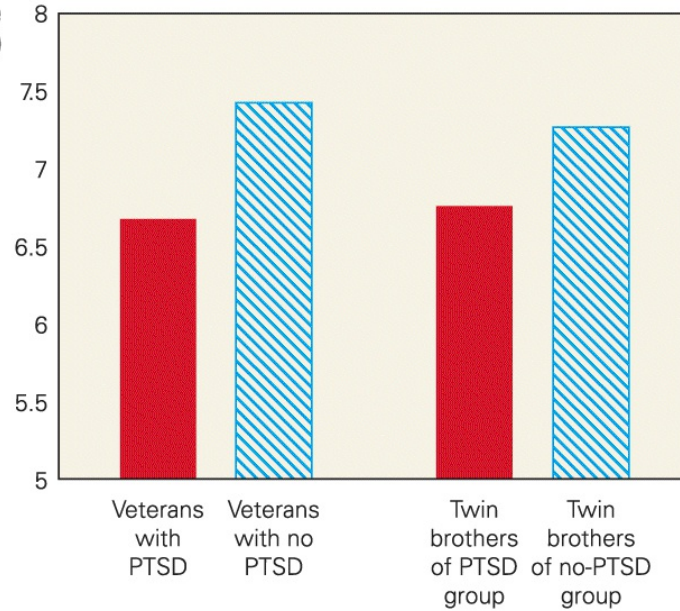
As illustrated in [Figure 10.16](#), the majority of individuals exposed to traumatic events do not develop PTSD. Apparently, preexisting vulnerability factors help determine an individual's risk for the condition, which is why one person will develop the disorder but another will prove resilient when exposed to the same traumatic event. Understanding these vulnerability factors is of critical importance because there is some evidence that interventions intended to prevent PTSD among individuals exposed to traumatic events can actually produce higher-than-expected

rates of the disorder—presumably by inducing symptoms in individuals who would most likely have been resilient if simply left alone to deal with the trauma in their own way ([Rothbaum & Davis, 2003](#)).

A better approach might be to give the preventive therapy only to those individuals who are most likely to develop PTSD. But how can we identify such individuals in advance? Structural MRI suggests some answers. Numerous studies have documented that individuals with PTSD typically have hippocampal volumes that are somewhat smaller than those of individuals who experienced similar trauma but did not develop PTSD ([Logue et al., 2018](#); [O’Doherty, Chitty, Saddiqui, Bennett, & Lagopoulos, 2015](#); [Smith, 2005](#)).

For instance, Mark Gilbertson and colleagues used brain imaging (MRI) to examine hippocampal volume in a group of veterans who had fought in the Vietnam War ([Gilbertson et al., 2002](#)). Even though the war officially ended in the 1970s, many veterans of that war still have trouble with combat-related PTSD symptoms. [Figure 10.17](#) (left) shows that the veterans who developed PTSD had a slightly but significantly smaller hippocampal volume—on average—than veterans who had not developed PTSD. One interpretation of these findings would be that PTSD might cause the hippocampus to shrink, since we know that long-term exposure to chronic stress can damage the hippocampus ([McEwen, 1997](#); [Sapolsky, 1996](#)). But another possibility is that the veterans who developed PTSD had a smaller hippocampus to start with—and that it made them more vulnerable to PTSD.

Hippocampal volume
(in cubic millimeters)



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

FIGURE 10.17 Hippocampal volume and PTSD Structural MRI from a group of combat veterans showed that, on average, those who had developed PTSD had smaller hippocampal volume than those who did not develop PTSD. The twin brothers of the veterans who had developed PTSD also had smaller hippocampal volume than the twin brothers of the veterans who did not develop PTSD. This suggests that the veterans with PTSD had smaller-than-average hippocampal volume and that this may have predisposed those individuals to develop PTSD when exposed to extreme stress such as wartime deployment and combat.

Data from [Gilbertson et al., 2002](#).

Description

The readings on the vertical axis range from 5 to 8 in increments of 0.5. The hippocampal volumes for different groups of people are as follows.

Veterans with PTSD-6.7, veterans with no PTSD-7.4, twin brothers of PTSD group-6.8, and twin brothers of no-PTSD-7.3.

Gilbertson and colleagues found a clever way to determine the answer: each of the veterans included in the study had an identical twin brother who had not fought in Vietnam and who had never developed PTSD. The twins of the veterans with PTSD had smaller-than-average hippocampal volumes, too ([Figure 10.17](#), right). This suggests that PTSD does not *cause* hippocampal shrinkage; rather, it seems that some individuals may have a smaller-than-average hippocampal volume that may *predispose* them to develop PTSD later ([Gilbertson et al., 2002](#)). For example, connections from the hippocampus to the prefrontal cortex are important in extinguishing learned associations; individuals with a slightly smaller hippocampus might be less

able to extinguish fear responses than their peers who have average hippocampal volumes. If an individual leads a relatively quiet life (like the brothers who were not sent into combat), this may not be a problem; however, if that same individual is exposed to extreme stress and trauma (like the brothers who did serve in the war), there could be heightened risk for PTSD.

Another brain area that has been implicated, unsurprisingly, is the amygdala. It has been known for years that individuals with PTSD often show increased amygdala responses to negative emotional stimuli, but—as with the hippocampus—it was hard to tell whether the abnormality was a preexisting condition or had developed after the trauma as a symptom of PTSD. The answer was discovered as a result of the Boston Marathon bombing in 2013.

In 2011–2012, researchers in the Boston area were performing a study using fMRI to study amygdala reactions in adolescents. Then, on April 15, 2013, two bombs went off near the finish line of the Boston Marathon. Three people were killed, more than 200 were injured, and thousands of Boston-area residents were told to “shelter in place” for several days during the subsequent hunt for one of the bombers. A month later, the researchers sent online surveys to the teenagers who had previously participated in the fMRI study, asking them about PTSD symptoms related to the attack. It turned out that those teenagers who had shown heightened amygdala reactions to negative emotional stimuli during the earlier fMRI study were at higher risk of developing PTSD in the wake of the bombing ([McLaughlin, Busso et al., 2014](#)). Thus, it appears that amygdala hyperactivity may precede, and confer risk for, PTSD in individuals later exposed to trauma.



David L. Ryan/The Boston Globe via Getty Images

In April 2013, two bombs went off near the finish line of the Boston Marathon, killing 3 people, injuring more than 200, and potentially traumatizing thousands of runners, spectators, and area residents. What factors might predict which individuals would develop PTSD as a result?

In addition to decreased hippocampal volume and heightened amygdala activity, other factors that make individuals vulnerable to PTSD may include certain genes ([Norrholm & Ressler, 2009](#)); increased startle responses to sudden, loud noises ([Orr et al., 2003](#); [Pitman et al., 2006](#)); and personality traits such as neuroticism, harm avoidance, and novelty avoidance ([Aidman & Kollaras-Mitsinikos, 2006](#); [Gil & Caspi, 2006](#); [Hatcher, Whitaker, & Karl, 2009](#)). On the other hand, individuals with strong social support from family and friends are somewhat *less* likely to develop PTSD after a traumatic event ([Pietrzak, Johnson, Goldstein, Malley, & Southwick, 2009](#); [Polusny et al., 2010](#)). Understanding the factors that make an individual vulnerable to PTSD or more resilient could be very useful in screening people for wartime service as well as for jobs such as firefighting, disaster recovery, and foreign aid work, where traumatic situations are especially likely to occur. Not all trauma is avoidable, of course; in fact, most people experience at least one traumatic event in the course of a lifetime. But knowing in advance which

individuals are most vulnerable to PTSD would provide the opportunity to help those individuals with targeted care and counseling if and when exposure to trauma occurs.

Interim Summary

- Certain psychological problems may involve emotional responses gone awry.
- Phobias are excessive fears that interfere with daily life. Specific phobias are those related to specific objects or situations, such as snakes, spiders, heights, or enclosed spaces. In agoraphobia, patients avoid leaving home or a “safe” environment for fear of having a panic attack in public.
- On the premise that some phobias arise through classical conditioning, the therapy of systematic desensitization uses the concept of extinction to reduce the ability of the phobic stimulus to elicit a fear response. Other phobias may arise through social transmission.
- In posttraumatic stress disorder (PTSD), a natural fear reaction does not subside with time, possibly reflecting a failure of extinction.
- Some research suggests that certain individuals have preexisting characteristics, such as reduced hippocampal volume or heightened amygdala response to emotional stimuli, that could increase the risk for PTSD following exposure to a traumatic event.

Synthesis

If you have ever tried to list the similarities between yourself and a rat, chances are that emotions did not top the list. Yet humans, chimpanzees, elephants, dogs, rats, and even the lowly *Aplysia* all show emotional responses. Confronted with the prospect of an electric shock, a rat may freeze and defecate, a sea snail may retract its siphon, and a dog may whine and put its tail between its legs—all overt behaviors and physiological changes that can be experimentally induced and measured. Moreover, emotional learning depends on similar brain substrates in many species, especially mammals, including the amygdala, the hippocampus, and the frontal cortex. This commonality allows the use of insights gained in rats and other nonhuman animals to help elucidate the neural bases of learned emotional responses in people.

As you saw in this chapter, the vast majority of research on emotions and learning has focused on a single emotion, fear. One reason is that the critical brain substrates for fear are at least partially understood, whereas the brain substrates for happiness and other positive emotions are only now being discovered. Another reason is that we can observe and measure fear responses, and therefore we can reasonably assume that anticipating shock leads to fear in animals. It has been less clear whether animals feel “sadness” or “surprise” and, if so, how we could induce and measure such emotions. But newer research focusing on animal models of emotions such as joy and disgust may lead to new breakthroughs in understanding the human versions of these emotions, too.

One more reason for the historical emphasis on fear is its association with clinical syndromes such as phobias, PTSD, and depression. Many researchers study fear in the hope that their findings will have clinical relevance. And indeed, existing clinical treatments such as systematic desensitization and virtual-reality therapy have been developed using our understanding of how emotional memories are acquired and maintained.

For example, our discussion of PTSD described research on the possibility of using drugs like propranolol that interfere with epinephrine to reduce an individual’s risk of developing PTSD. The idea, in the aftermath of a trauma such as the 9/11 terrorist attacks, would not be to make the survivors forget the traumatic episode but rather to reduce the damaging emotional reactions to the memory. Existing studies do not show a reliable benefit of propranolol injections in reducing rates of PTSD, but even if the treatment did work reliably, it would raise serious ethical

questions. Should such prophylactic treatment be routinely administered to individuals who experience a trauma, despite the fact that the majority of such individuals will never develop PTSD if simply left alone? And, on a more fundamental level, if people routinely altered the emotional content of their “negative” episodic memories, would the result be happier lives for all, or would it be a colorless existence in which nothing really matters because nothing causes emotionally charged memories? Without unpleasant memories, would we lose some of our ability to learn from mistakes—the very reason emotions may have evolved in the first place?

Chapter 10 Review

Know Your Key Terms

adrenaline

amygdala

arousal

autonomic nervous system (ANS)

Cannon–Bard theory of emotion

cognitive expectancy

conditioned avoidance

conditioned emotional response

conditioned escape

cortisol

depression

emotion

epinephrine

fear response

fight-or-flight response

flashbulb memory

glucocorticoid

James–Lange theory of emotion

learned helplessness

mood congruency of memory

norepinephrine

optogenetics

phobia

piloerection

posttraumatic stress disorder (PTSD)

somatic theories of emotion

stress

stress hormone

systematic desensitization

tend-and-befriend response

two-factor theory of avoidance learning

Quiz Yourself

1. Emotion involves three distinct but interrelated phenomena—namely, _____, _____, and _____—that are produced in response to an affecting situation. ([p. 418](#))
2. The _____ theory of emotion states that conscious feelings of emotion occur after the mind senses the physiological responses associated with arousal. The _____ theory of emotion states that conscious feelings of emotion and bodily responses (physiological responses and overt behaviors) occur simultaneously and independently. The _____ theory of emotion states that a combination of cognitive appraisal and perception of biological changes together determine our experience of emotion. ([pp. 421–423](#))
3. The _____ is a collection of nerves and structures that control internal organs and glands and help mediate the physiological components of emotion. ([p. 420](#))
4. A(n) _____ is an excessive and irrational fear of an object, a place, or a situation. One type of therapy for conditions of this kind includes _____, in which successive approximations of the fear-evoking stimulus are presented while the patient learns to remain relaxed. ([pp. 453, 455](#))
5. The _____ response (also known as _____) is a collection of bodily responses, including increased blood flow to the muscles, increased respiration, and depressed digestion and immune functions, that prepare the organism to deal with a threat either by facing it down or by running away. ([p. 420](#))
6. The principle that it is easiest to retrieve memories that match our current mood or emotional state is known as _____. ([p. 435](#))
7. The _____ is a brain area critical for learning and expressing emotional responses as well as for mediating the emotional modulation of memory. ([pp. 440–441](#))

8. In _____, animals learn to make particular responses in order to terminate an aversive stimulus. In _____, animals learn to make particular responses in order to prevent exposure to that aversive stimulus. ([p. 430](#))
9. When we experience emotions such as fear or anger, the ANS releases _____ that help mediate the fight-or-flight response. Two examples are _____ and _____. ([p. 420](#))
10. _____ is a psychiatric condition that involves feelings of sadness as well as a general loss of initiative and activity. Some think it reflects a type of conditioning called _____, in which exposure to an uncontrollable punisher teaches an organism that its responses are ineffective, which in turn reduces the motivation to attempt to escape. ([p. 433](#))
11. A memory formed under conditions of extreme emotion and that seems especially vivid and long-lasting is called a(n) _____. ([p. 436](#))
12. _____ theories of emotion are based on the central premise that physiological responses to stimuli come first, and they determine or induce emotions. ([p. 422](#))
13. The _____ is a cluster of physiological, motor, and conscious reactions that accompany the emotion of fear. An example of a physiological reaction is _____, where a mammal's hair stands on end, making the animal look bigger and more threatening than it is. ([pp. 418, 425](#))
14. Any stimulus or event that causes bodily arousal is a form of _____. ([p. 420](#))
15. _____ is a psychological disorder that can develop after exposure to a horrific event, with symptoms including _____, _____, _____, and _____. ([p. 456](#))
16. The laboratory technique called _____ is used to make neurons responsive to light, such that light stimulation can be used to turn those neurons "on" and "off" at will. ([p. 444](#))
17. The _____ theory of avoidance learning holds that avoidance learning involves an interaction between classical conditioning and operant conditioning. An

alternate theory—the _____ theory—holds that animals choose among possible responses based on the expected outcome of each response. ([pp. 431–432](#))

18. Although epinephrine cannot cross the blood–brain barrier, the related neurotransmitter _____ can enter the brain and affect brain function. ([p. 449](#))
19. A _____ is a classically conditioned response (CR) that is produced in response to a CS that has been paired with an emotion-evoking US. ([p. 429](#))
20. Some individuals may respond to threat by protecting offspring and seeking out social support, a strategy known as _____. ([p. 426](#))

Concept Check

1. [Chapter 3](#) defines habituation as decreased responding to a repeatedly presented stimulus. Do you think habituation is an example of emotional learning? Why or why not?
2. After losing a game with a lopsided score, professional football teams are statistically more likely to perform worse than usual in their next game. This tendency is particularly pronounced if the upcoming opponent is considered hard to beat ([Reisel & Kopelman, 1995](#)). How might this phenomenon be explained in terms of the principles of emotional learning?
3. Decorticate animals (animals with their cortex surgically removed) often show “sham rage,” overreacting to seemingly trivial stimuli as though they were being seriously threatened. Why might this happen?
4. Suppose you have a roommate who is terrified of spiders, to the point of being unable to take a shower if there is a spider in the bathtub unless you are there to kill the spider or remove it. Based on what you read in this chapter, speculate about why your roommate has this fear.
5. Continuing the example in question 4, what might you suggest to help your roommate overcome this fear?

Answers to Test Your Knowledge

Theories of Emotion

1. James–Lange: Janet sees and hears the band; her body experiences arousal, including increased heart rate and pupil dilation; as a result of this arousal, she experiences conscious feelings of joy and excitement.
2. Cannon–Bard: Janet sees and hears the band. In response to these stimuli, she feels joy and excitement, and, at the same time, her body experiences arousal.
3. Two-factor theory: Janet sees and hears the band, which causes arousal; in the context of a music concert, cognitive appraisal allows Janet to interpret this arousal as signaling joy and excitement.

Brain Substrates of Emotion

1. The elevator ride may be a CS that activates Jared's amygdala; outputs from the central nucleus travel to the ANS and turn on the fight-or-flight response.
2. Jared's current fear response acts as a contextual stimulus and helps the hippocampus and/or cortex trigger memories of earlier events that caused a fear response. If the diving incident was traumatic enough, Jared may even have a flashbulb memory for that event.
3. Rationally, Jared knows he won't fall off the observation deck. His prefrontal cortex, the site of higher decision making, helps to overrule the impulse to flee.
4. Appreciation of the beautiful view is itself an emotional response that can be encoded by the hippocampus as part of an episodic memory for this day. If Jared is lucky, the positive emotions will strengthen encoding of that memory. Maybe the next time he's at the top of a tall building, the context will serve as a retrieval cue, helping to trigger the memory of his pleasure at the view from the top of the Space Needle, and this may help counteract some of his fear.

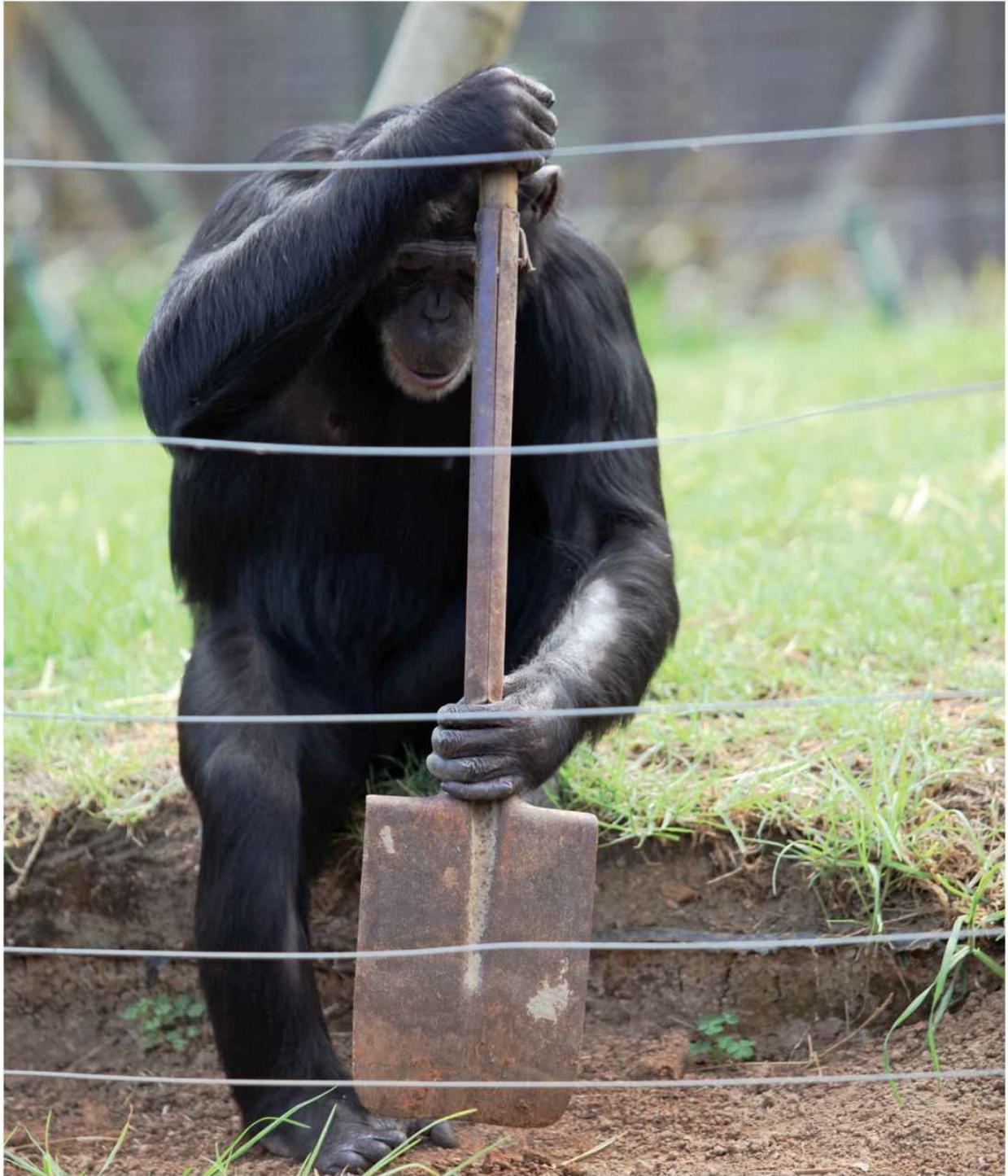
Classical Conditioning and Phobias

In this example, the US is the panic attack. Because Nancy experienced a panic attack at the grocery store, the grocery store has become a CS that can itself evoke a CR—her fear of another panic attack.



Chapter 11 Social Learning and Memory

Observing, Interacting, and Reenacting



Matthew Tabaccos/Barcroft/Getty Images

Behavioral Processes

[Copying What Is Seen](#)

[Copying What Is Heard](#)

Learning and Memory in Everyday Life: Karaoke

Social Transmission of Information

Learning and Memory in Everyday Life: Learning What to Like from Super Bowl Ads

Brain Substrates

Identifying Mirror Neurons

Mirror Neurons in Songbirds

Clinical Perspectives

Imitative Deficits After Stroke

Autism Spectrum Disorder

DOLPHINS ARE THE ONLY KNOWN animals other than humans that can imitate both actions and sounds on request: scientists have trained dolphins to imitate human actors as well as computer-generated sounds when instructed to do so. Chimpanzees and orangutans can imitate other animals' actions when humans ask them, but they seem unable to imitate sounds. Parrots can imitate sounds but usually require lots of experience hearing the sound first; unlike humans and dolphins, parrots have not shown the flexibility needed to imitate a novel sound the first time they hear it. Most other animals show no ability to imitate actions or sounds when instructed to do so by humans. The reason could be that they don't know how to imitate, don't have the right vocal control mechanisms, or have not been properly trained to imitate; or it may be that, unlike dolphins and apes, they don't understand human instructions.

Yes, you read that last statement correctly. Scientists can communicate with dolphins and apes, and these newly discovered communication channels have revealed unsuspected learning and memory abilities in those animals. One dolphin, named Akeakamai (pronounced "uh-KAY-uh-ka-MY," Hawaiian for "lover of wisdom"), learned to interpret hand signals asking her to imitate the actions of dolphins and humans. She learned this skill through one-on-one interactions with her human instructors. She would watch intently as a hairless ape stood in front of her, gesticulating rapidly with its oddly shaped "flippers." Over time, she learned that one of the gestures meant she was supposed to imitate a recently demonstrated action, such as a human spinning around or a dolphin shooting a basketball through a hoop. When Akeakamai interpreted this gesture correctly, the instructor rewarded her with shouts of approval. Occasionally, the instructor's vocal accolades proved infectious, and Akeakamai joined in the celebration with her own loud vocalizations. Akeakamai observed the actions of those around her and reacted based on her past training, her recent experiences, and her emotional state.

Social interactions and observations can contribute to learning and memory in many ways. You can watch television to learn about exotic vacation destinations, listen to the radio to learn about an approaching storm, or read a textbook to learn about how your brain enables you to learn and remember. People generally assume that humans learn much of what they know by *interacting* socially with others, *observing* how things work, and then *reenacting* what they have observed. Elementary school teachers, for example, often demonstrate how tasks should be performed (e.g., showing how to multiply two numbers) and then encourage students to repeat the demonstrated steps in class. Many people believe that the cognitive abilities such feats require make human learning and memory different from that of all other animals. However, this view is based more on presumption than on experimental evidence. Here, we explore what psychologists have discovered about how and what humans and other animals learn through social interactions and observations.

The mechanisms of social learning have been studied less extensively than most of the other learning and memory processes described in this book. As a result, the basic behavioral processes and neural substrates involved remain uncertain, and theoretical explanations of how social interactions contribute to learning are more speculative.

11.1 Behavioral Processes

The term **social learning** refers to situations in which an individual actively monitors events or other phenomena involving other individuals and then chooses later actions based on those observations. You are currently observing (reading) the words in a textbook describing learning and memory, and you may use your observations to guide your later behavior when you are studying for and taking tests on the subject. If your test includes short-answer questions, you might even answer those questions with sentences very similar to the ones you are reading in these pages. In fact, the more precisely you replicate what you have read (or heard during lectures) when answering such questions, the more likely it is that your professor will judge your answers to be correct. Successful performance on written tests depends heavily on social learning. Social learning is one of the many means by which an individual may learn and is much broader than just learning how to behave in social situations. (In other words, social learning is not to be confused with learning to socialize.)

social learning

Learning from others through observation or communication.

Social learning differs from classical and operant conditioning in that researchers cannot reliably predict what an organism will learn from observing the actions of others or the consequences of those actions. One reason it is difficult to predict how observations will affect future behavior is that there is no way for one observer (say, a researcher) to detect what another observer (e.g., a research subject) actually learns from observing ongoing events without probing the observer's knowledge. Generally, most social learning is *latent* (see [Chapters 1](#) and [3](#) for further discussion of latent learning) and may not become evident except under particular circumstances. An organism can observe a wide variety of events without showing any obvious changes in behavior or giving any other indication of what has been observed or of how the impressions the organism received from those observations are affecting it. If you watch your fellow students during a lecture, do you think you will be able to tell if one of them is daydreaming about being on the beach?

Psychologists are frequently faced with this uncertainty. Edward Thorndike, for example, permitted untrained cats to “observe” the actions of other cats as the latter demonstrated their knowledge of how to escape from a puzzle box. On this basis, he concluded that cats could not imitate other cats in order to learn how to escape from the box ([Thorndike, 1898](#)). But there is no

way to know what Thorndike's "observer" cats focused on when they were given the opportunity to watch other cats escaping from boxes. Perhaps the food placed as a reward outside the box or the identity of the cat trapped in the box was more interesting to the feline observer than the specific actions the cat inside the box was performing.

Another reason it is difficult to predict how observations will affect future behavior is that nothing compels an observer to copy any given model. Furthermore, even if the observer does copy a model's actions, there is more than one learning mechanism that can lead to matching actions, as we now discuss.

Copying What Is Seen

Social learning differs from most other forms of learning discussed in this book in that not all species can learn by reproducing what they observe. In fact, many psychologists and anthropologists believe that only humans learn extensively by copying others. **Copying** consists of replicating what one observes another doing; this is generally what people mean when they use the term "imitation." Children *can* learn a great deal by copying the actions of adults and other children. However, there is very little experimental data showing what children *do* learn through imitation in everyday life.

copying

The act of doing what one observes another organism doing.

Most psychologists' views about the role of copying in learning stem from influential experimental studies conducted in the early 1960s by Albert Bandura. Bandura (b. 1925) grew up in Canada before heading to the University of Iowa for graduate school. Soon after starting his academic career at Stanford University, he became interested in how parents' aggressive behavior might affect the actions of their children. Bandura and his colleagues wanted to see whether preschool children would become more aggressive after observing aggressive adults ([Bandura, Ross, & Ross, 1961](#)). The researchers had one group of children observe adults beating up a "Bobo doll" (an inflatable clown doll), while other groups of children (control groups) simply played in an empty room or observed adults playing quietly with toys. Afterward, Bandura used a one-way mirror to spy on the children as they played in a room containing the Bobo doll ([Figure 11.1](#)). Children who had observed an adult pummeling the Bobo doll were more likely than children in the control groups to pound on the doll themselves; and, most

importantly, their attack styles were often similar to those used by the adults. Bandura and colleagues concluded that the children had learned new aggressive actions by observing the actions of an adult.



Courtesy Albert Bandura

FIGURE 11.1 Scenes from a Bobo doll experiment After viewing an adult acting aggressively toward a Bobo doll, some children imitated what they had seen ([Bandura et al., 1961](#)). The pictures have been paired to emphasize similar actions.

Description

Panel 1: A woman is pulling punches at a Bobo doll in a dim-lit room. Panel 2: The woman lifts and throws the Bobo doll in the dim-lit room. Panel 3: The woman knocks the doll with a hammer. Panel 4: The woman kicks the Bobo doll and sends it flying in the air. Panel 5: A little girl child pins down the Bobo doll. Panel 6: A little boy lifts the Bobo doll. Panel 7: The boy swings a hammer at the Bobo doll. Panel 8: The little girl kicks the doll. The last four panels that show similar actions are placed below the first four panels.

Studies like Bandura's Bobo doll experiment seem to suggest that children will copy aggressive acts they have observed. However, the children were tested in a context similar to the one in which they observed the aggression occurring, soon after they viewed the aggressive acts. It is unclear whether they would have behaved similarly if they had encountered a Bobo doll at their neighbor's house. In addition, the children in Bandura's experiment who showed significant copying of aggressive acts had first been provoked by being deprived of an attractive toy immediately before the test ([Bandura et al., 1961](#)). Specifically, the experimenter told the provoked children that some attractive toys in the room were there for them to play with, but as soon as a child began playing with a toy, she told the child that these were her very best toys and that she had decided to reserve them for some other children. Children who had viewed an

aggressive model but who had not been provoked were actually less likely to behave aggressively during the test than were children who did not observe any aggressive acts. This finding suggests that viewing aggressive acts can in some cases inhibit aggressive behavior rather than increase it.

One of the main findings from the Bobo doll experiments was that viewing an adult acting aggressively toward an inflatable toy strongly influenced the later behavior of children presented with that same toy, despite the fact that the children had never been reinforced nor punished for performing such actions. The absence of direct reinforcement or punishment would appear to exclude the possibility that the children were learning Bobo attack styles through operant conditioning, which requires that the learners' actions be either reinforced or punished. Bandura proposed that children observed the actions of the adult, formed ideas about what actions could be performed using the doll, and later used memories of those ideas to reproduce the adult's actions ([Bandura, 1969](#)).

Note that by this account, the formation of memories that changed behavior occurred while the children were observing the adult, *not* when they were copying the behavior. In other words, what children encoded was information about a specific episode, and imitative acts were based on children's memories of that episode. The children's imitative acts simply revealed what the children had learned from watching someone demonstrate novel actions. Bandura called these demonstrations **modeling**; he considered social modeling to be a prerequisite for all kinds of copying.

modeling

Demonstration of actions.

Social Learning Theory

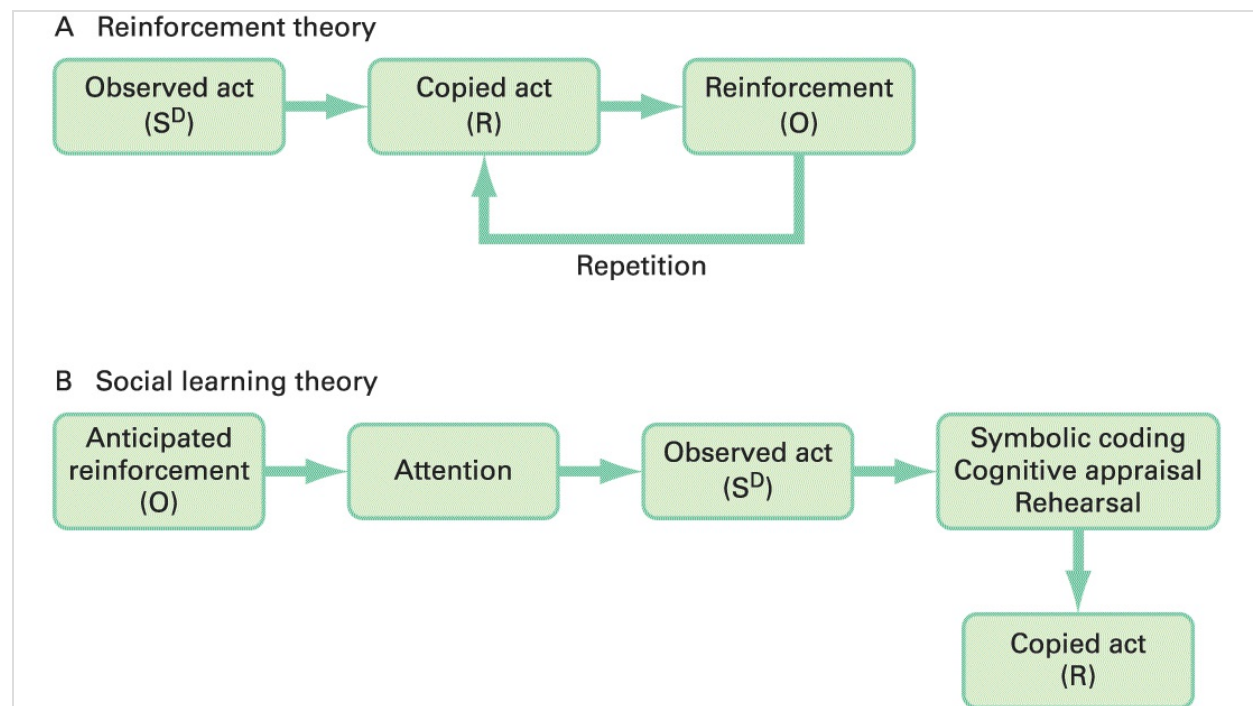
To account for cases of social learning such as the imitative walloping revealed in Bandura's Bobo doll experiments, psychologists developed a broad theory of human behavioral development called **social learning theory**. A key feature of social learning theory was (and, for some adherents, still is) the idea that the kinds of reinforcement an individual has observed or experienced in the past will determine how that individual will act in any given situation. Early social learning theorists proposed that reinforcement determines personality traits ([Rotter, 1954](#)) and that social learning is a special case of operant conditioning (the topic of [Chapter 5](#)), in

which actions that replicate observed acts are either directly or indirectly reinforced ([Miller & Dollard, 1941](#)).

social learning theory

A theory of human behavior, prominent from the 1940s through the 1960s, proposing that the kinds of reinforcements an individual has experienced in past social contexts will determine how that individual will act in any given situation.

According to [Bandura \(1969\)](#), observers can gain information about whether a particular action will be rewarded or punished by witnessing the outcomes of a model's actions. A person who copies actions that have been seen to lead to positive outcomes may in turn experience positive outcomes ([Figure 11.2A](#)). If so, the person has a greater likelihood of again copying such actions in the future (consistent with Thorndike's law of effect).



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FIGURE 11.2 Social learning theory (A) In traditional theories of operant conditioning, actions are conditioned through repeated experiences in which behaviors are either reinforced or punished. (B) Social learning theory suggests that an event observed once can create a memory that may change how an individual will behave in the future before that behavior has been reinforced or punished. An observer viewing a scene may anticipate that certain actions observed in the scene could lead to some reward on a later occasion. Later on, attention to and appraisal of ongoing events generates memories that make copying of previously observed actions possible.

Information from Bandura, 1971.

Description

Illustration A depicts the Reinforcement theory. Observed act (S superscript D) leads to Copied act (R) which when led to a reinforcement (O) results in the repetition of the copied act (R).

Illustration B depicts the Social learning theory. Anticipated reinforcement (O) develops attention, which produces an observed act (S superscript D). This leads to symbolic cognitive appraisal rehearsal that eventually brings out a copied act (R).

Modern social learning theory places less emphasis on conditioning and instead explains behavior in terms of more cognitive processes, such as evaluating possible future outcomes and learning about how people interact ([Figure 11.2B](#)). A basic premise of contemporary social learning theory is that any behavior can be learned without direct reinforcement or punishment ([Bandura, 1986](#); [Ladd & Mize, 1983](#)). This assumption is critical to claims that exposing children to violent images in games or movies (without providing direct reinforcement) can increase the likelihood of the children engaging in violent acts at a later date. We consider such claims in more detail later in this chapter.

Certainly, expectations of reinforcers and punishments will influence the likelihood that a learned action will be performed, but modern social learning theory argues that the learning itself (the knowledge gained about an action and its outcome) is the result of observation rather than the result of conditioning. In other words, a relationship between an action and an outcome need only be observed, not performed, in order to be learned. Within this framework, a copied action is simply a performance that reveals what has been learned (as noted earlier for children who copied adults in the Bobo doll experiment).

Bandura cited four basic processes to explain how people copy what they see. First, the *presence of a model* is thought to increase an observer's attention to the situation. The actions of others can be especially salient cues that act as a magnet for attention ([Bandura, 1986](#)). Second, memories for the observed situation must be stored in an *accessible format* so that they can guide later actions. If the observer forgets how an action was performed, it will be difficult for that person to copy the action. Third, the observer must have the *ability to reproduce the action*. You might remember quite well what it looks like when someone dunks a basketball, but unless you can jump quite high, you will not be able to copy this action. Finally, the observer must have some *motivation for reproducing* the observed actions. You probably would not burn your money just because you saw (or heard about) someone else doing it, even though it would be

easy to do.

In some cases, the status or identity of the model can provide the motivation for an observer to imitate an action. For example, individuals are more likely to imitate the actions of someone they admire. Similarity between a model and the observer also increases the likelihood of copying, especially if the outcome is desirable. In other cases, the desirability of the observed outcome itself is enough to provide motivation for an imitative act. For instance, you might be more likely to burn your money if you observe that other people who burn their money are invited to parties with celebrities.

Copying can involve either the replication of actions that have been observed or the performance of novel actions that lead to the observed outcome of a modeler's actions ([Morgan, 1896](#)). For example, when Iam was still in elementary school, he happened to see his parents picking berries and putting them in a bucket. A few days later, he copied his parents' berry-picking technique but put the berries in his pockets rather than in a bucket. The movements Iam made while picking berries replicated those of his parents. Copying in which motor acts are replicated is called **true imitation**. The movements Iam performed to store the berries that had been picked, however, differed from those of his parents. But Iam's and his parents' actions led to a similar outcome: berries were collected in a container. Copying that reproduces an outcome without replicating specific motor acts is called **emulation**. In both of these cases of copying, the core processes that make Iam's socially learned novel actions possible are his ability to recall a past episode and his ability to transform visual percepts into action plans.

true imitation

Copying that involves reproducing motor acts.

emulation

Copying that involves replicating an outcome without replicating specific motor acts.

Studies of True Imitation: Replicating Actions

Scientists use the term "true imitation" to distinguish a certain kind of copying from the many phenomena that people assume are examples of imitation but that do not actually involve any copying of actions ([Thorndike, 1898](#); [Thorpe, 1963](#); [Whiten, Horner, Litchfield, & Marshall-Pescini, 2004](#)). Several examples of such "faux imitation" are provided later, under the heading "[Matching Actions Without Truly Imitating](#)."



JGI/Jamie Grill/Getty Images

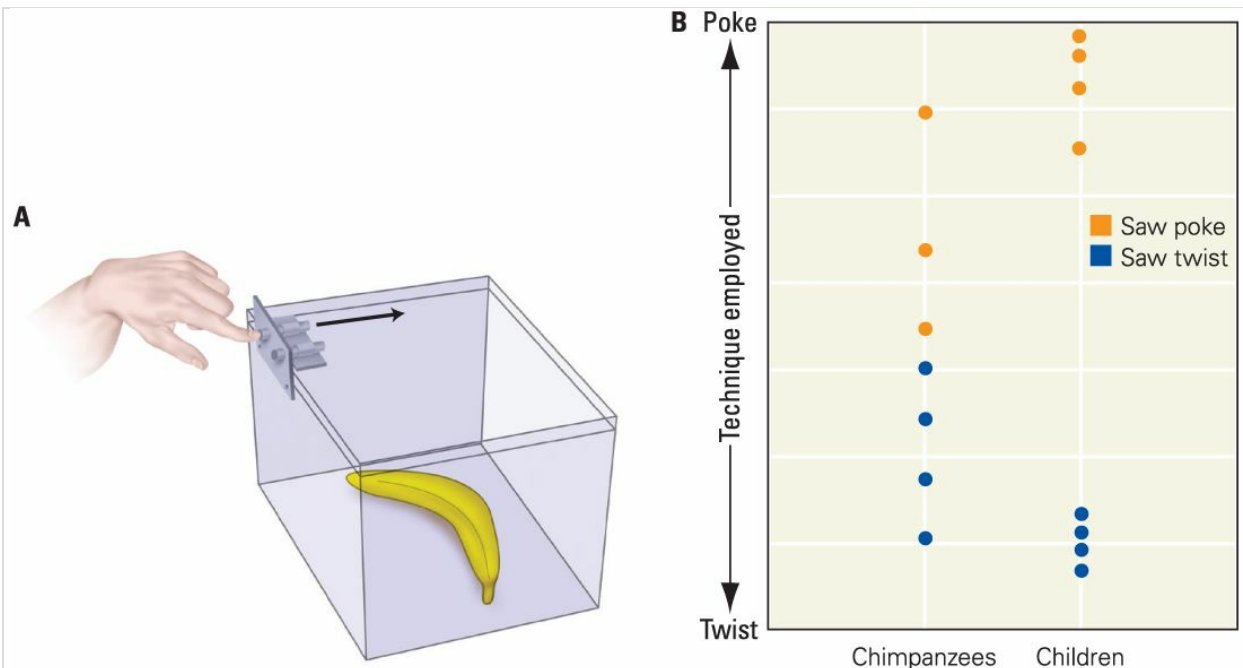
Is this an instance of true imitation?

One technique that has been developed to investigate true imitation abilities is called the **two-action test**. In preparation for this test, two individuals are each trained to perform a different action, but the two actions have the same outcome. For instance, one individual might be trained to dislodge an object that is stuck by hitting at it with a stick, and the other might be trained to pry the object free with a stick. Next, one group of naïve observers is allowed to watch a demonstration of one of the techniques, and a second equally naïve group is allowed to observe the other technique. If the naïve observers later perform the operation in a way that matches the technique they observed, then their behavior is accepted as evidence of true imitation.

two-action test

A technique developed to investigate imitation abilities; it involves exposing naïve animals to demonstrators trained to achieve the same goal using different actions.

The two-action test has enabled researchers to compare imitative capacities across species. For example, one experiment compared true imitation in young children (ages 2–4) and juvenile chimpanzees (ages 4–5) ([Whiten, Custance, Gomez, Teixidor, & Bard, 1996](#)). In this study, children and chimpanzees were shown how to open a plastic box to obtain a food reward ([Figure 11.3A](#)). Half of the participants saw an adult human open the box by poking pins out of its latch before lifting the lid. The other half saw an adult human open the box by twisting and pulling the pins out of the latch. After observing their respective models, the participants were given a chance to open the box themselves.



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers
 Research from Whiten et al., 1996.

FIGURE 11.3 Two-action test of true imitation in children and chimpanzees (A) Children and chimpanzees observed an adult human use one of two techniques (poke or twist) to open a plastic box containing a reward. (B) Later, both children and chimpanzees were more likely to use the box-opening technique they had observed than the one they had not observed. This means that both chimps and children can truly imitate.

Research from [Whiten et al., 1996](#).

Description

A. The illustration shows a human hand twisting the lever to open a box containing a banana.

B. The horizontal axis of the scatter plot is labeled chimpanzees and children. The vertical axis shows the technique employed: Twist toward the bottom half and poke toward the upper half. The graph shows that the number of chimpanzees and children who saw twist were more than those who saw poke. Chimpanzees saw four twists and three pokes. Children saw four twists and four pokes.

Nothing about this task requires precise copying because the box can be opened in many ways that are equally effective. Nevertheless, both children and chimpanzees usually copied the opening technique that they had observed ([Figure 11.3B](#)). Children were more likely than chimpanzees to copy the details of the observed actions, including details that were not essential to getting the box open. Two-action tests have been used to study copying in several species, including birds ([Akins & Zentall, 1996](#); [Dawson & Foss, 1965](#)), rodents ([Heyes & Dawson,](#)

1990), and primates ([Whiten et al., 1996](#)), and in most of these studies, evidence of true imitation was found.

Humans can copy a wide variety of actions, including novel, nonfunctional actions like beating a Bobo doll. To assess whether any other animals share the ability to truly imitate nonfunctional actions requires methods that are more flexible than the two-action test or the technique used by Bandura and colleagues. One approach involves teaching animals to copy actions on command (often referred to as the “do-as-I-do” task). For instance, researchers trained a chimpanzee to copy a person’s actions when the person gave the command “Do this!” ([Hayes & Hayes, 1952](#)). The researchers would perform an action, such as clapping hands, while telling the chimpanzee to “Do this!” and then would record whether or not the chimpanzee performed the same action. Initially, the chimpanzee was unlikely to copy the actions the first time they were demonstrated. However, after learning to “Do this!” for about 12 actions, the chimpanzee was able to replicate certain new actions the first time she was asked.

In a replication of this experiment, two other chimpanzees showed that they could replicate 30 actions the first time they saw them ([Custance, Whiten, & Bard, 1995](#)). This is the same type of instruction that the dolphin Akeakamai learned in order to copy actions on request, although she learned to respond to gestures rather than speech.

One reason psychologists have gone to such lengths to precisely define true imitation and to develop definitive methods for identifying the phenomenon is to test the claim of some researchers that true imitation requires high-level cognitive processes that are unique to humans ([Bandura, 1986](#); [Piaget, 1962](#)). For example, true imitation by humans is often taken to imply some awareness on the observer’s part that he or she is voluntarily copying actions.

Perspective taking, or imagining oneself in the place of another, is another cognitive ability that some researchers have suggested is a prerequisite for the voluntary copying of actions. It is an ability that few species other than humans may possess. Perspective taking is thought to facilitate true imitation because it enables people (or certain other animals) to copy others without watching themselves doing so. For instance, you can reproduce someone’s facial expressions without watching yourself make faces. Consistent with this idea, individuals with autism spectrum disorder who have problems with perspective taking show a reduced ability to copy

observed actions. (The imitative deficits associated with autism are discussed further in [Section 11.3.](#))

perspective taking

Imagining oneself in the place of another.

Your ability to imagine an outside observer's perspective provides feedback that may help you copy novel actions, but that does not mean that true imitation always entails perspective taking. In fact, imagining the perspective of the individual performing an action may lead you to adopt a totally different copying strategy that is more effective than merely reproducing the action you observed.

Studies of Emulation: Reproducing Outcomes

As noted earlier, emulation involves reproducing an observed outcome without replicating the actions the model used to achieve that outcome. Chimpanzees and adult humans often perform actions that lead to the same outcome as another individual's actions rather than copying the specific motor acts observed. For example, chimpanzees that saw people using a rake and conventional raking motions to collect food outside their cage were more likely than other chimpanzees to use a rake for the same purpose, but they did not reliably copy the specific raking actions they observed ([Nagell, Olguin, & Tomasello, 1993](#)). For instance, a chimpanzee might hold the metal end of the rake and swing the wooden handle to knock the food closer to the cage. This type of copying has been distinguished from more exact copying, and some theorists have claimed that when specific actions are not replicated, the copying does not qualify as true imitation ([Tomasello, Davis-Dasilva, Carnak, & Bard, 1987](#); [Wood, 1989](#)).

Most of the evidence of emulation in chimpanzees and adult humans comes from studies intended to show the degree to which subjects are capable of true imitation. For example, in the plastic-box study described previously, the human children were more likely to truly imitate the precise box-opening actions of the model (e.g., poking the pegs with an index finger), whereas the chimpanzees were more likely to emulate the behavior of the model while using their own methods for opening the plastic box (e.g., pushing the pegs with their palms). One interpretation of this difference is that the chimpanzees learned some features of the task by observing the model's behavior but then chose actions different from the ones they had observed in order to perform the task. Interestingly, when adult humans were tested on the plastic-box task, they

performed more like chimpanzees in that they were more likely to emulate than to precisely copy the model's actions ([Horowitz, 2003](#)).

Emulation is sometimes considered to be a less sophisticated mechanism of social learning than true imitation is, in part because emulation can occur in nonsocial contexts ([Hopper, Lambeth, Schapiro, & Whiten, 2015](#)). For instance, “ghost displays,” in which an observer sees objects moving and functioning without any operator present (think self-driving cars), can enable the observer to achieve similar outcomes through emulation.

Emulation is not the only form of “nonsocial” social learning. The following discussion reviews several other processes that can contribute to social learning without requiring the kinds of complex cognitive mechanisms associated with true imitation and with social learning theory.



*“You’ve just been copying things from my
sex diary into your sex diary.”*

Peter C. Vey/The New Yorker Collection/The Cartoon Bank

True imitation? Emulation? Both?

Description

The cartoon shows a woman holding a diary saying to a man, open quotes You’ve just been copying things from my sex diary into your sex diary. Close quotes

Matching Actions Without Truly Imitating

Although much of social learning research has focused on organisms copying the actions of others, there are a number of ways that humans and other animals can replicate the actions of others without employing true imitation. Carefully executed experiments are often necessary to determine which instances of action matching involve true imitation and which do not, because to the casual observer, many of these other phenomena look just like true imitation. Below, we describe three such phenomena that closely resemble true imitation: emotional contagion, observational conditioning, and stimulus enhancement. Each of these processes qualifies as copying because it involves replicating actions that others were observed performing, and each can contribute to social learning. But all three are thought to involve less sophisticated mechanisms than those involved in true imitation.

Emotional Contagion

When you hear laughter, you too may feel the urge to laugh (which is why laugh tracks are a common feature of sitcoms). When you see someone yawn, you will be more likely to yawn. When one baby starts crying on a plane, other babies on the plane are likely to join in. The inborn tendency to react emotionally to sights or sounds of emotion in other members of one's species is called **emotional contagion** ([Byrne, 1994](#); [Kramer, Guillory, & Hancock, 2014](#)). Emotional contagion is a relatively common phenomenon, in which an observer's detection of a given emotional response increases the likelihood that the observer will produce a similar response. Typically, the kinds of actions that lead to emotional contagion are not actions that an animal or a person has learned. Babies yawn and cry in utero. The actions triggered by emotional contagion usually match precipitating actions but *not* as a result of imitation; the matching reaction is instead an unconditioned response (like those described in [Chapter 4](#)).

emotional contagion

An inborn tendency to react emotionally to visual or acoustic stimuli that indicate an emotional response by other members of one's species, typically in ways that replicate the observed response.

Observational Conditioning

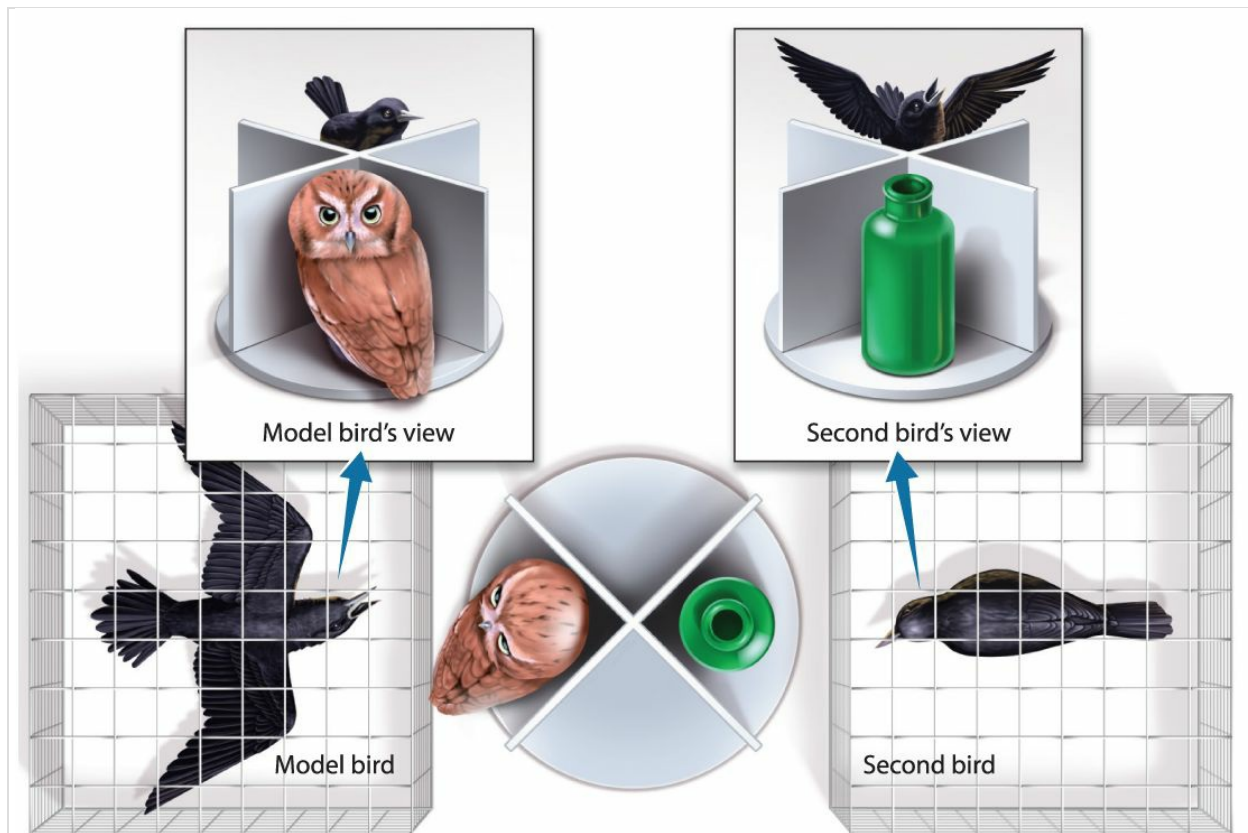
We describe a situation in [Chapter 10](#) in which naïve lab monkeys learned to fear snakes after watching wild monkeys react fearfully to them ([Mineka & Cook, 1988](#)). In this case, observer monkeys experienced emotional contagion when they saw the fearful responses of wild monkeys. They associated their unconditioned fear (sparked by the wild monkeys) with the

snake (a conditioned stimulus). As described in [Chapters 4 and 10](#), this is exactly the sort of association that is learned during classical conditioning. Instances such as this, in which an individual learns to associate an emotional response with a previously neutral stimulus after observing others act emotionally when that stimulus is present, have been described as **observational conditioning** ([Heyes, 1994](#)). This is one way that phobias can develop (discussed further in [Chapter 10](#)).

observational conditioning

A process in which an individual learns an emotional response after observing similar responses in others.

Some instances of observational conditioning are almost indistinguishable from situations that have been described as imitative learning. In fact, early social learning theorists once used this similarity to argue that all social learning experiments involve nothing more than classical or operant conditioning. Consider the following example. In the wild, blackbirds automatically attack predators when they observe other blackbirds attacking those predators (an unconditioned response). Once a blackbird has learned from such experiences that a particular object is worthy of attack, it will continue to attack this object on sight for quite a while: the object has become a conditioned stimulus that evokes a conditioned response (attack). In laboratory studies of this phenomenon, one blackbird can “teach” a second bird to attack something as harmless as a plastic bottle ([Curio, Ernst, & Vieth, 1978](#)). Researchers get the birds to do this by showing the “model” bird a stuffed owl, a predator that blackbirds naturally attack. Meanwhile, the second bird observes the model’s attacks, but a clever optical illusion causes the second bird to see a bottle under attack rather than an owl ([Figure 11.4](#)). Consequently, the second blackbird learns to attack bottles.



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FIGURE 11.4 Observational conditioning in blackbirds Blackbirds can learn to attack harmless objects such as bottles if they observe other blackbirds doing so—or are tricked into thinking that is what they are observing, as is shown here.

Information from [Shettleworth, 1998](#).

Description

The illustration shows a model bird and a second bird in cages placed on either side of a circular disc with partitions resembling a partitioned tray with four standing compartments that hold a stuffed owl and a plastic bottle in two compartments. A stuffed owl is placed on the partitioned tray facing the model bird, and a plastic bottle is placed on the opposite side of the owl facing the second bird. The model bird is shown flapping its wings and about to attack while the second bird remains calm. Another two illustrations are shown above. The first illustration labeled Model bird's view shows a stuffed owl and a calm black bird behind. The second illustration, labeled Second bird's view, shows a bottle and a black crow flapping its wings from behind.

This might seem like a clear case of true imitation: one bird sees another bird attacking a bottle and then does the same thing. Moreover, this experiment is a lot like the Bobo doll experiment described earlier, which is considered by many to be a definitive demonstration of true imitation by children. The key difference is that in the case of the blackbirds, attacking possible predators

is a species-specific reflex that almost always occurs when a blackbird either detects a predator or sees another blackbird attacking something; both cases are unconditioned stimuli that evoke the same unconditioned response. In this example of social learning, the observing blackbird is being observationally conditioned: the attacking “model” bird is an unconditioned stimulus—its initial attacks lead to an unconditioned attack response by the observer—and the bottle is the conditioned stimulus.

The fact that the bottle-attacking response can be interpreted as “merely” classical conditioning doesn’t prove that birds are incapable of social learning or true imitation. In fact, researchers have conducted numerous studies demonstrating social learning in ravens and crows ([Bugnyar & Kotrschal, 2002](#); [Holzhaider, Hunt, & Gray, 2010](#)). The bottle-attacking study simply illustrates the difficulty of identifying the mechanisms that contribute to social learning and true imitation in natural situations.

Stimulus Enhancement

Observational conditioning and emotional contagion are two phenomena in which an observer copies the actions of others but without truly imitating those actions. A third phenomenon that bears a deceptive resemblance to true imitation is an observer’s attention being drawn to stimuli by the actions of others. Redirecting an individual’s attention can lead to matching actions that may be mistaken for imitation ([Roberts, 1941](#); [Thorpe, 1963](#)). For example, you may have seen a group of people (typically younger people) collectively directing their attention to try to trick other people into looking up at the sky. The way this trick generally works is that the group will start looking up at the sky and pointing as if there were something interesting (or frightening) happening up there. Inevitably, a passerby will look up to where the group is looking to see what all the excitement is about. The passerby then experiences confusion because there is nothing to see.



imagewerks Japan/Getty Images

Stimulus enhancement consists of directing attention to a particular part of the environment; this can lead to actions that appear similar to imitation. In this case, do you think the stimulus being enhanced is an object or a particular location?

Because the passerby replicates the looking actions of the tricksters, one might think that the passerby is imitating them. In this case, however, the similarity in actions is a by-product of a similar focus of attention. Just because people watching a movie in a movie theater behave similarly does not mean that they are all imitating one another. Direction of one organism's attention toward specific objects, events, or locations within an environment as a result of another organism's action is called [stimulus enhancement](#) ([Spence, 1937](#)).

stimulus enhancement

A process in which observations of other individuals draw an organism's attention toward specific objects, events, or locations within an environment.

Stimulus enhancement can powerfully affect what an individual learns ([Heyes, 1994](#)).

Essentially, the individual's focus is drawn to a subset of features of the environment that might provide more useful information than other features present. In general, stimulus enhancement increases the likelihood that an individual will be exposed to particular stimuli and their associated consequences. Individuals may learn things after being attracted to the same locations and objects as other individuals, but they do so without ever needing to reenact any observed actions. Moreover, like emulation, stimulus enhancement does not require a visible model; thus,

a spotlight works well to direct the audience's attention to a particular location on a stage.

Given the many ways that individuals can learn from others, you might wonder whether it is common for social learning to involve an actual reenactment of actions observed in the past. There are no definitive studies showing how frequently true imitation occurs. However, one case in which this form of social learning seems to play a dominant role is during early language learning by children. The following section describes how reproducing sounds differs from visually based imitation, as well as some ways that vocal imitation abilities may contribute to social learning of communicative skills (a topic explored further in [Chapter 12](#)).

TEST YOUR KNOWLEDGE

Is It True Imitation?

Much of what people describe as imitation may not be what scientists consider to be true imitation and in fact might not even involve copying. Below are some scenarios that may seem to, and possibly do, involve true imitation. Your job is to try to provide alternative explanations for what is happening. ([Answers appear at the end of the chapter.](#))

1. You smile at your friend's baby. The baby smiles back. Is your friend's baby imitating you?
2. You discover that almost anything you do makes the baby smile (apparently, you are a talented clown). But when you stick out your tongue at the baby, the baby sticks out her tongue at you. Is she imitating you?
3. Iam sees his father make a face when he is eating asparagus, and his father's expression seems to indicate that he thinks asparagus is disgusting. Later in the week, Iam's grandmother serves asparagus and notices that Iam is making a similar face while eating. Is Iam imitating his father?
4. While watching *America's Funniest Home Videos*, you see a video of a cat jumping up to turn a doorknob so as to open the door and go outside. Is the cat imitating people?
5. Iam has stumbled into one of the oldest frustrations of childhood. He tells his friend, "Stop repeating whatever I say!" His friend replies, "Stop repeating whatever I say!" Iam counters, "I mean it, you better stop!" To which his friend replies, "I mean it, you better stop!" And so on. Is Iam's friend imitating him?

Copying What Is Heard

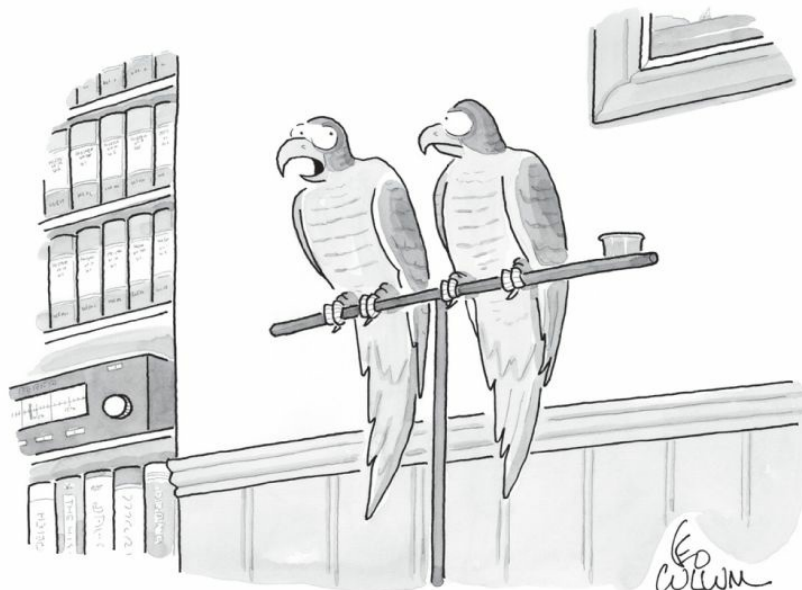
The one feature of human learning and memory that most seems to set humans apart from other animals is the role that language plays. The foundation of language and speech acquisition is social learning. In particular, the ability to produce sequences of sounds that others can understand is critical for language use by most humans. This ability depends on recognizing and replicating specific sounds.

Early psychologists considered the fact that some birds copy the sounds of other animals to be

the strongest evidence that animals other than humans have the ability to remember and reenact events in the same way as humans ([Morgan, 1896](#); [Romanes, 1898](#)). [Thorndike \(1911\)](#) argued, however, that birds do not truly imitate sounds but instead learn through trial and error to make sounds similar to ones they hear, a special case of conditioning. Modern researchers tend to agree with Thorndike, rejecting the idea that vocal imitation is truly imitative ([Byrne & Russon, 1998](#); [Heyes, 1994](#)). As illustrated earlier, it can be quite tricky to tell when an animal is really imitating observed actions. Of course, Thorndike never had the opportunity to watch home videos of parrots swearing on YouTube. You really only have to see one parrot telling someone to “f*** off” to suspect that perhaps birds *can* truly imitate human speech.

Vocal Imitation: Reproducing Sounds

When infants are learning to talk, they sometimes replicate features of speech produced by others around them ([Kuhl & Meltzoff, 1996](#)). For example, infants with normal hearing raised in different locales produce speech sounds similar to the ones they hear, whereas infants who are deaf do not. In one study, infants 12 to 20 weeks old who listened to an adult making vowel sounds immediately produced sounds similar to those they heard ([Kuhl & Meltzoff, 1996](#)). This implies that speech-imitation abilities are present very early in development.



“That’s not my political opinion. That’s just stuff I hear on the radio.”

Leo Cullum/The New Yorker Collection/The Cartoon Bank

Speech imitation is a type of [vocal imitation](#), copying that involves pushing air through vocal

membranes to re-create some properties of a previously experienced sound. This action may be either voluntary (as is seen in professional impersonators) or involuntary (as occurs when you unconsciously hum a catchy song). In many ways, vocal imitation is like copying an action you have never seen. When you imitate someone's speech, you don't ever see the motor actions the person used to produce that speech. You only hear the outcomes of those actions (which some have argued makes vocal imitation a kind of emulation). Nevertheless, to reproduce features of the speech you heard, you need to produce motor acts similar to the ones the model used when speaking, a key criterion for true imitation.

vocal imitation

Copying that involves vocal reproduction of sounds.

Surprisingly, no mammals other than humans are able to reproduce speech sounds as well as birds can. In fact, most mammals, including our closest ape relatives, show little or no ability to vocally imitate and rarely learn to produce vocalizations through experience. Dolphins are the only mammals other than humans that are known to be able to truly imitate sounds ([Janik & Slater, 1997](#); [Mercado, Mantell, & Pfordresher, 2014](#)). Some dolphins, like Akeakamai, have been trained to vocally imitate computer-generated sounds on command ([Richards, Wolz, & Herman, 1984](#)) and may also spontaneously copy sounds ([Hooper, Reiss, Carter, & McCowan, 2006](#); [Reiss & McCowan, 1993](#)). Sometimes when Akeakamai was instructed to vocally imitate a sound that she could not easily reproduce, she would produce a transposed copy of the sound ([Richards et al., 1984](#)), much as a singer might transpose a song to a higher- or lower-pitch range to make it easier to sing. (See "[Learning and Memory in Everyday Life](#)" for a brief discussion of how vocal imitation abilities contribute to people's prowess at karaoke.) Several other marine mammals also show some vocal imitation abilities, including humpback whales and belugas ([Ridgway, Carder, Jeffries, & Todd, 2012](#)).

In contrast to mammals, numerous species of birds have the ability to vocally imitate. Some, such as the superb lyrebird, can precisely replicate a wide variety of human-made sounds (including car alarms and chainsaws) with astonishing precision. The ability of infants or young birds to vocally imitate adults may help the youngsters learn new communication skills, but to a certain extent, the vocal imitation is separate from the learning. For instance, it is relatively easy to construct an electronic toy parrot that can reproduce sounds, yet such mimicking machines show little if any capacity to learn to communicate. It is only when true imitation guides

communicative skill learning that the advantages of imitative abilities in communication are revealed.

LEARNING AND MEMORY IN EVERYDAY LIFE

Karaoke

Karaoke is a relatively recent form of musical entertainment in which amateurs attempt to sing popular songs along with recorded instrumental music. Such performances have been compared with singing in the shower but with an audience and less soap. Unlike sing-alongs, in which singing is typically led by someone with at least moderate musical talent, karaoke is pretty much a free-for-all in which singing ability is optional. Some participants in karaoke produce sounds so awful and out of tune that it is hard to believe they actually think their performance counts as singing.

Surprisingly, little is known about what it takes for someone to produce a good karaoke performance. Obviously, you have to be familiar with the song you are attempting to sing, so memory for the tune and the way the words correspond to the notes is a crucial ingredient. This alone is not sufficient, though, because plenty of people are familiar with songs such as “Happy Birthday” and yet still sound like broken robots when they attempt to sing them. Recent experiments suggest that the secret ingredient to singing pop songs like a star may be good vocal imitation abilities ([Pfordresher & Brown, 2007](#)). A person might have a nearly perfect memory of what the original song sounds like, but without the ability to translate that memory into vocal actions that re-create those sounds, this memory will be of little help.

The good news is that people tend to underestimate their ability to vocally imitate songs. Only about 15% of college students are actually hopeless at karaoke, even though many college students (about 60%) believe their ability to sing melodies familiar to them is quite poor ([Pfordresher & Brown, 2007](#)). Perhaps as more is learned about how vocal imitation works, even those who sound like dying cows can be transformed into songbirds.

Learning to Vocalize Without Imitating

Animals that do not naturally copy vocalizations appear to be unable to learn this skill. Early in the twentieth century, researchers made great efforts to encourage chimpanzees to vocally imitate speech. Catherine Hayes described the most successful effort of the time, in which a chimpanzee named Viki eventually learned to garble “cup,” “mama,” and “papa” ([Hayes, 1951](#)). This achievement provides no evidence of vocal imitation abilities in chimpanzees because Viki was explicitly trained to produce words through reinforcement and physical molding of her lip positions.

You have probably seen videos of dogs and cats that seem to say “I love you” or “I want my mama.” Many animals can be trained to produce speech-like sounds, but these again are examples of conditioning (usually involving shaping, as described in [Chapter 5](#)), not vocal imitation. As noted above, [Thorndike \(1911\)](#) claimed that vocal imitation is not true imitation

(reenactment of previously observed actions) but a specialized form of conditioning in which individuals gradually adjust the sounds they make by monitoring how their performance differs from sounds they have previously heard. Differences between produced sounds and remembered sounds provide feedback that in principle could help an individual gradually make sounds more similar to ones previously heard.

Learning to vocalize by adjusting one's own sound production based on sounds one has heard is called **vocal learning**. Some researchers suggest (like Thorndike) that vocal imitation is a type of vocal learning. However, adult humans vocally imitate sounds in many situations where they are not really learning anything new, such as at weddings, when the bride and groom are instructed to repeat specific vows, or when a beatboxer imitates the sound of a drum machine at the couple's reception. Vocal imitation can contribute to vocal learning, as it does in young children learning to speak, but these two phenomena are not identical. All animals that can vocalize are capable of vocal learning, but only a few learn to produce sounds through vocal imitation. A dog may learn to bark when its owner says "Speak!" or to whine more loudly to go outside when a TV is on (both instances of vocal learning), but no dog has ever learned to copy novel sounds like dolphins can. **Table 11.1** compares and contrasts vocal learning with vocal imitation as well as with the other types of social learning that you have read about so far.

TABLE 11.1 Comparison of social learning processes

Sight based	Sound based
True imitation: Observer replicates visible body movements.	Vocal imitation: Observer replicates audible sounds.
Emulation: Observer achieves same outcome as a model.	
Observational conditioning: A neutral stimulus becomes a conditioned stimulus.	Vocal learning: Vocal responses are gradually modified based on feedback.



James D. Watt/SeaPics.com

Humpbacks are the only mammals other than humans that naturally learn to sing songs by hearing them—a unique case of vocal learning. Or is it a case of vocal imitation?

vocal learning

Modifying one's vocal outputs on the basis of sounds one has heard.

Genes and Memes in Song Learning

The most extensively studied form of vocal learning in any species (including humans) is song learning by birds. Birdsongs typically consist of multiple sounds separated by regular silent intervals and last from 1 to 10 seconds. The timing, order, and qualities of individual sounds in the songs are all components that a singing bird can control. Birds that learn their songs do so in part by listening to other birds. Young songbirds use their memories of songs produced by mature birds to guide their own song production; these memories can last a lifetime.

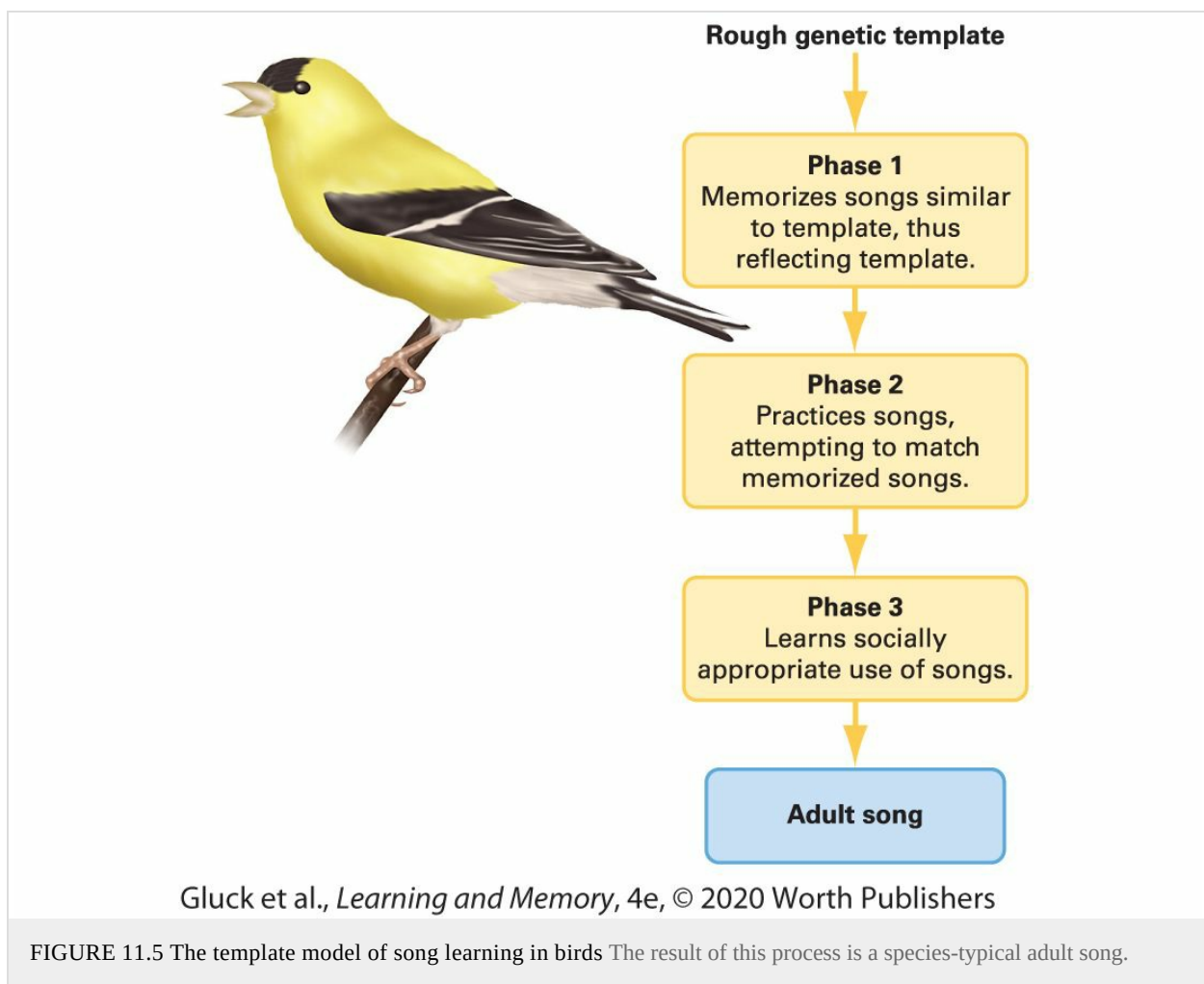
By isolating individual songbirds at a very young age (even prior to birth) so that they never hear another bird singing, scientists have been able to prove that songbirds do learn their songs socially, from each other. Singing insects, frogs, and monkeys that are isolated early in life will develop relatively normal songs ([Bradbury & Vehrencamp, 1998](#)). In contrast, many songbirds never sing normally if they are isolated early in development ([Hinde, 1969](#)).

Song learning seems to occur in three phases. To begin with, most songbirds appear to be born with a crude inherited “template” that biases their brain toward storing certain sound patterns. In the first phase of song learning, young birds memorize the songs they hear that best fit their genetically defined template; these memorized songs provide a model against which later performance is judged. Young birds exposed to both the songs of their own species and the songs of other species will typically only learn to produce the song associated with their species ([Marler, 1997](#)). Similar constraints seem to operate in human infants learning a spoken language. Human infants are exposed to a wide range of sounds, including the sounds of birds, dogs, and musical instruments. Yet most of the vocalizations they first learn to produce are speechlike sounds. This could be because speech sounds are inherently easier for infants to produce or because, like songbirds, infants are genetically predisposed to recognize and reproduce particular sounds.

For birds, the second phase of song learning ensues when the young bird begins attempting to sing songs. While the bird is singing, it hears itself singing and can compare its own song with memories of songs it has heard in the past. With practice, the young bird increases the match between its own performance and the songs it remembers hearing. In the third stage of song learning, the bird learns when to sing. For example, a territorial songbird might sing primarily when it hears a song it has never heard before or when it hears a familiar song sung by an

unfamiliar bird. The actions and reactions of a nearby female may also influence a male songbird's singing.

These three phases of song learning are described in, and collectively make up, the [template model of song learning](#) (Figure 11.5). Note that the template model of song learning overlaps with social learning theory in that young birds use memories of observed events to guide their actions at a later time. The process of song learning can potentially involve vocal imitation, but, as noted by Thorndike, gradually modifying how sounds are produced (vocal learning) does not necessarily require reenacting any previously experienced event.



Description

The illustration shows an American Goldfinch perched on a slender branch to the left and the template model to the right. The model is as follows. Rough genetic template. Phase 1, memorizes songs similar to template, thus reflecting

template. Phase 2, practices songs, attempting to match memorized songs. Phase 3, learns socially appropriate use of songs. adult song

template model of song learning

A model of song learning that has three basic phases: song memorization, song practice, and song utilization.

Like human speech, the songs that birds sing may contain differences in *dialect*, the locale-specific idiosyncrasies of a given language ([Baptista & King, 1980](#); [Marler, 1970](#); [Thorpe, 1958](#)). Just as your accent can reveal to others where you grew up and learned to speak, a bird's "accent" can reveal where the bird grew up and learned to sing. Baby birds raised by an adult of another species can sometimes even learn the foreigner's song ([Immelman, 1969](#)). Interestingly, if young birds are simply exposed to recordings of foreign songs, they do not learn to sing them ([Pepperberg, 1994](#)). The young birds need to be able to see the singer in order to learn the foreign song. Both dialects and foreign-song learning further show that some birds learn what to sing based on past experiences.

Some birds continue to change the songs they sing even as adults ([Lynch, Plunkett, Baker, & Jenkins, 1989](#)). For example, a new bird may join a community and start singing a song none of the resident birds know. Members of the community may then add that new song to their repertoire. The process is similar to the spread of a meme on the Internet in that once a few birds start singing the new song, it is not long before many birds pick it up. The next section examines how such cultural interactions can contribute to social learning.

Social Transmission of Information

Imagine that you are waiting to purchase a soda from a vending machine, and you notice that the person ahead of you just lost money in the machine. On the basis of observing the person's misfortune, you might decide not to put your money into that particular machine. This is an example of **social transmission of information**, a process in which an individual directly or indirectly communicates information that may affect the future actions of group members. Social transmission of information is seen in all human cultures. It is historically evident in the development of spoken and written language, and more recently in the development of libraries, television, telephones, and the Internet. Through these various channels, information can be transmitted rapidly to vast numbers of individuals. Note that social transmission of information can lead to social learning (e.g., if someone tells you not to drink the tap water in a foreign

country) but often may not (as would likely occur if someone were to tell you right now that you are reading).

social transmission of information

A process in which an individual directly or indirectly communicates information that may affect the future actions of group members.

The many ways in which humans are able to transmit information enables them to learn more rapidly than all other animals in an almost unlimited range of contexts. Unfortunately, this phenomenon has seldom been studied in the laboratory, except in relation to skill learning. For example, [Chapter 8](#) describes an experiment in which a person was asked to kick a target as rapidly as possible. His performance changed incrementally, in keeping with the *power law of practice*, improving quickly at first and then more slowly (see [Figure 8.2B](#)). However, when he was shown a film of someone kicking the target in a more effective way, his performance improved dramatically. In this particular experiment, information about better ways to kick was socially transferred by means of a film. The person in this experiment probably improved by copying the model he visually observed, but because the experiment focused on skill learning rather than on true imitation, the exact mechanism of learning was not closely assessed. Coaches may similarly have athletes view video footage of their own or their opponents' performances to help the athletes refine their skills.

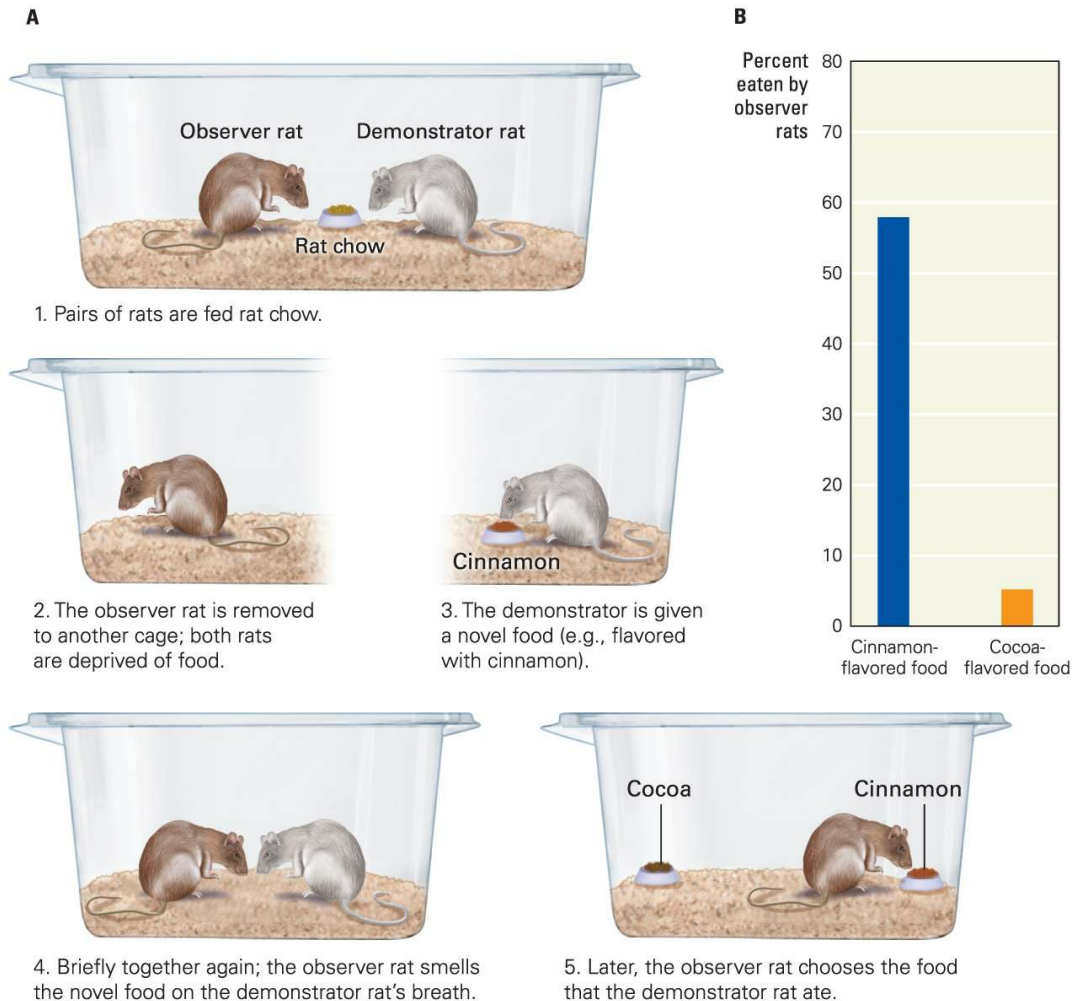
Because so much of the transfer of information between humans takes place in highly sophisticated ways, it is easy to overlook examples of socially transmitted information in other species. However, the very complexity of social transmission of information in humans makes it harder for researchers to understand how the process works. Studies of simpler forms of information transfer that can be measured in other species are thus important for identifying the mechanisms underlying social transmission of information.

Learning Through Social Conformity

If you've ever looked through your parents' or grandparents' photo albums, you have probably come across one or more pictures that made you wonder how they could ever have gotten it into their heads to wear clothes that were so ugly or uncomfortable. You may even have found a picture of the younger you wearing clothes and a hairstyle that you would be humiliated to be seen in today. In contrast, if you have seen photos of tigers taken in the 1920s, you would be

hard-pressed to tell them apart from photos of tigers that were taken yesterday. Why is it that humans do things to collectively change their appearance over time? Why do people even cut their hair or shave at all?

Studies of the social transmission of food preferences in rats may shed some light on such questions ([Galef & Wigmore, 1983](#)). Infant rats begin life preferring foods their mother has eaten because they can taste those foods in her milk. However, rats naturally pay attention to the food consumption of other rats and can learn to eat novel foods after observing that another rat has eaten them. In studies of this phenomenon, rats are typically housed together in pairs for a few days, during which they are fed rat chow ([Figure 11.6A](#)). One rat from each pair—chosen to be the “observer” rat—is moved to another cage. *Both* rats are deprived of food for a day, and then the “demonstrator” rat is fed cinnamon- or cocoa-flavored food (half of the demonstrator rats are given one flavor and half the other flavor). Each observer rat is then returned to its original cage and allowed to interact with the demonstrator rat (that has remained in the cage) for 15 minutes. On the subsequent day, the observer rats are given access to two food bowls, one containing cocoa-flavored food and the other cinnamon-flavored food. Observer rats typically eat more food of the flavor they smelled on the demonstrator’s breath ([Figure 11.6B](#)). This shows that observer rats acquired information about foods from the demonstrator rats and later used this information when choosing food.



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FIGURE 11.6 Social transmission of food preferences by rats Given a choice of two novel foods, observer rats are more likely to eat the food they smelled on a demonstrator rat's breath. This means that information about food has been transmitted between the rats.

Information from [Shettleworth, 1998](#).

Description

The illustration labeled a shows the follows,

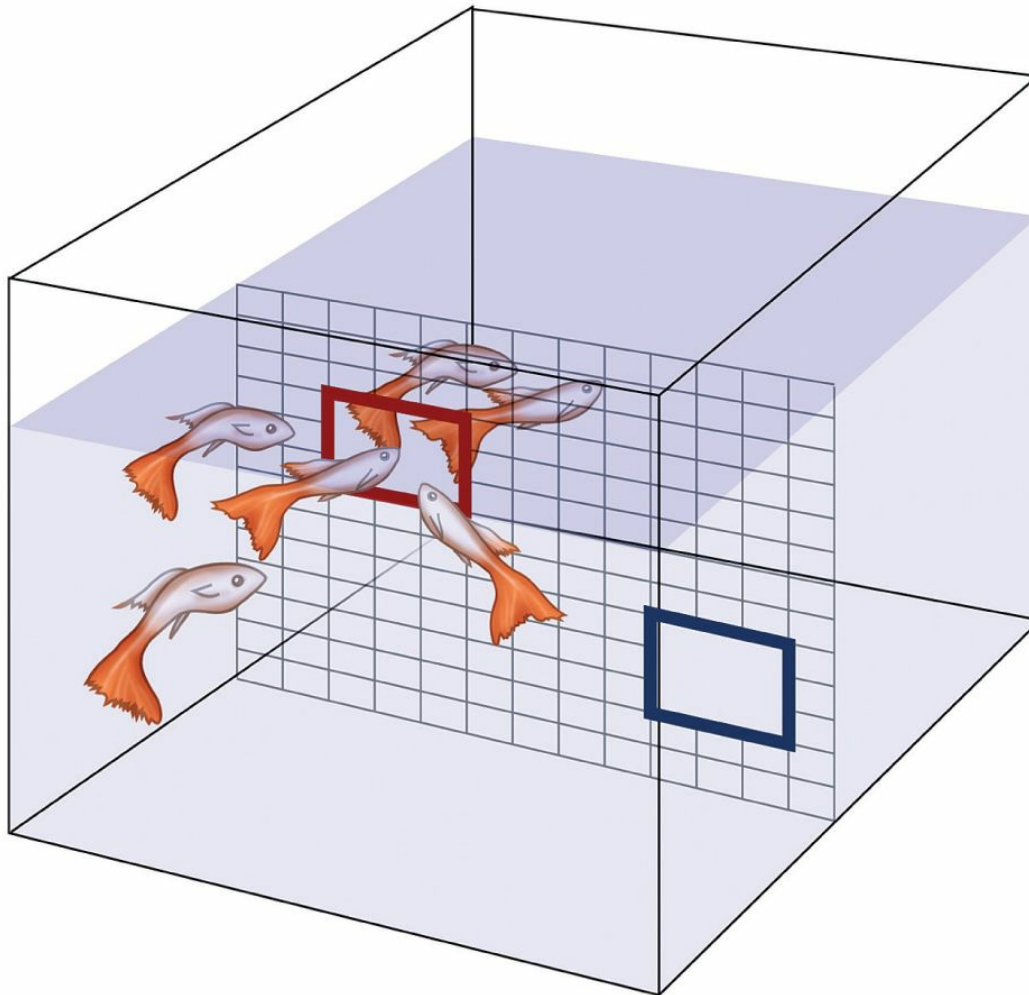
1. Pairs of rats are fed rat chow. The image shows two rats and a bowl of rat chow in a cage.
2. The observer rat is removed to another cage. Both rats are deprived of food.
3. The demonstrator is given a novel food (e.g., flavored with cinnamon). The accompanying image shows a rat and bowl of food flavored with cinnamon.
4. Briefly together again. The observer rat smells the novel food on the demonstrator rat's breath. The accompanying image shows the two rats close to one another inside a cage.

5. Later, the observer rat chooses the food that the demonstrator rat ate. The accompanying image shows the cage with observer rat sitting close to the bowl of cinnamon-flavored food while the bowl of cocoa-flavored food sits beside.

The graph labeled b shows the food types, Cinnamon-flavored food, and Cocoa-flavored food, on the horizontal axis and percent eaten by observer rats on the vertical axis. The data of the graph are as follows, Cinnamon-flavored food, 58 percent. Cocoa-flavored food, 5 percent.

Observer rats need to smell food in the context of another rat's breath to acquire a new food preference ([Galef, 1996](#)). Once a food becomes preferred, that preference may be transmitted to successive generations. Several factors affect how easily food preferences are transmitted to others ([Galef & Whiskin, 1997](#)). For example, the likelihood of transfer is increased if the observer views larger numbers of demonstrations or if those demonstrations are performed by larger numbers of demonstrating rats. Isolated (one-time) observations produce only a short-term effect, but repeated exposures lead to long-term retention.

In general, rats tend to follow what the majority of colony members do; this tendency to adopt the behavior of the group is called **social conformity**. Social conformity has many protective functions, but it can also hinder the development of novel behavior patterns that might be advantageous. In one experiment, guppies (a kind of fish) were trained to follow specific escape paths in order to evade an artificial predator ([Brown & Laland, 2002](#)). The artificial predator consisted of a net being moved across a tank to trap the guppies, and the escape paths were holes at two different locations in the net ([Figure 11.7](#)). One of the holes was closed during training of demonstrator guppies, and those guppies quickly learned where the one open hole in the net was situated. After training, the demonstrator fish continued to use only the escape path they had learned, even after the second hole was opened.



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FIGURE 11.7 Social transmission of escape routes by guppies After demonstrator guppies were trained to escape from a net by swimming through a particular hole (with a second escape hole closed), observer guppies tended to use the same escape path as the demonstrator guppies when both holes were open.

Research from [Brown & Laland, 2002](#).

Description

The illustration shows a cuboid tank with a net partitioning it into two. The net has two rectangular openings, one on the upper left corner and one on the lower right. All the guppies escape to the other side of the net through the opening on the upper left corner.

social conformity

The tendency to adopt the behavior of the group.

When naïve fish were introduced into this situation, with both holes open, they typically chose to

escape through the hole that they observed the trained fish using. If the demonstrator guppies were then removed from the tank, the observer guppies continued to use the hole they had seen the demonstrator guppies using, even when the alternate path led to a quicker escape. These results from rats and fish suggest that social conformity may be driven in part by stimulus enhancement: that is, odors, locations, or actions that are relevant to one individual become more salient to others.

Social conformity allows rapid acquisition of information that may be adaptive for a particular situation, but it can lead to traditions that may provide no general benefit. Interestingly, a reduction in the number of trained guppies (i.e., a decrease in the number of demonstrators during training) increased the likelihood that new escape solutions would emerge ([Brown & Laland, 2002](#)). Apparently, even guppies can be affected by peer pressure.

Social Manipulation of Behavior

When Iam entered elementary school, his parents (like most other parents) began worrying about how much time he spent watching television and about what he would learn if he watched programs they considered inappropriate. He didn't watch any more TV than the average child (about 24 hours per week), but this still seemed like a lot to Iam's parents. In particular, they worried that commercials might be making their son overly materialistic and that trailers for action movies glamorized violent behavior. Their worries intensified when Iam reached adolescence and began copying what they considered to be the "bizarre" behaviors of his YouTube idols. Like most other parents, Iam's parents worried about what he would learn from observing and copying the behaviors he encountered through mass media.

Researchers have collected strong evidence of a general association between violent behavior and increased exposure to violent media ([Anderson et al., 2003](#)). For example, the amount of violent TV watched by children in elementary school is correlated with their aggressiveness as teenagers and with their criminal behavior as adults ([Anderson et al., 2003](#)). Similarly, statistics show the rate of homicides to have increased dramatically soon after television was introduced in the United States, whereas no such increases were observed in regions where television was banned during the same time period ([Centerwall, 1992](#); see [Figure 11.8](#)). Given that such correlations exist, the main questions relevant to our discussion of social learning processes are (1) Are observations of violent media a direct cause of aggressive behavior? And, if so, (2) To

what extent does exposure to violent media lead individuals to copy violent actions that they have observed?

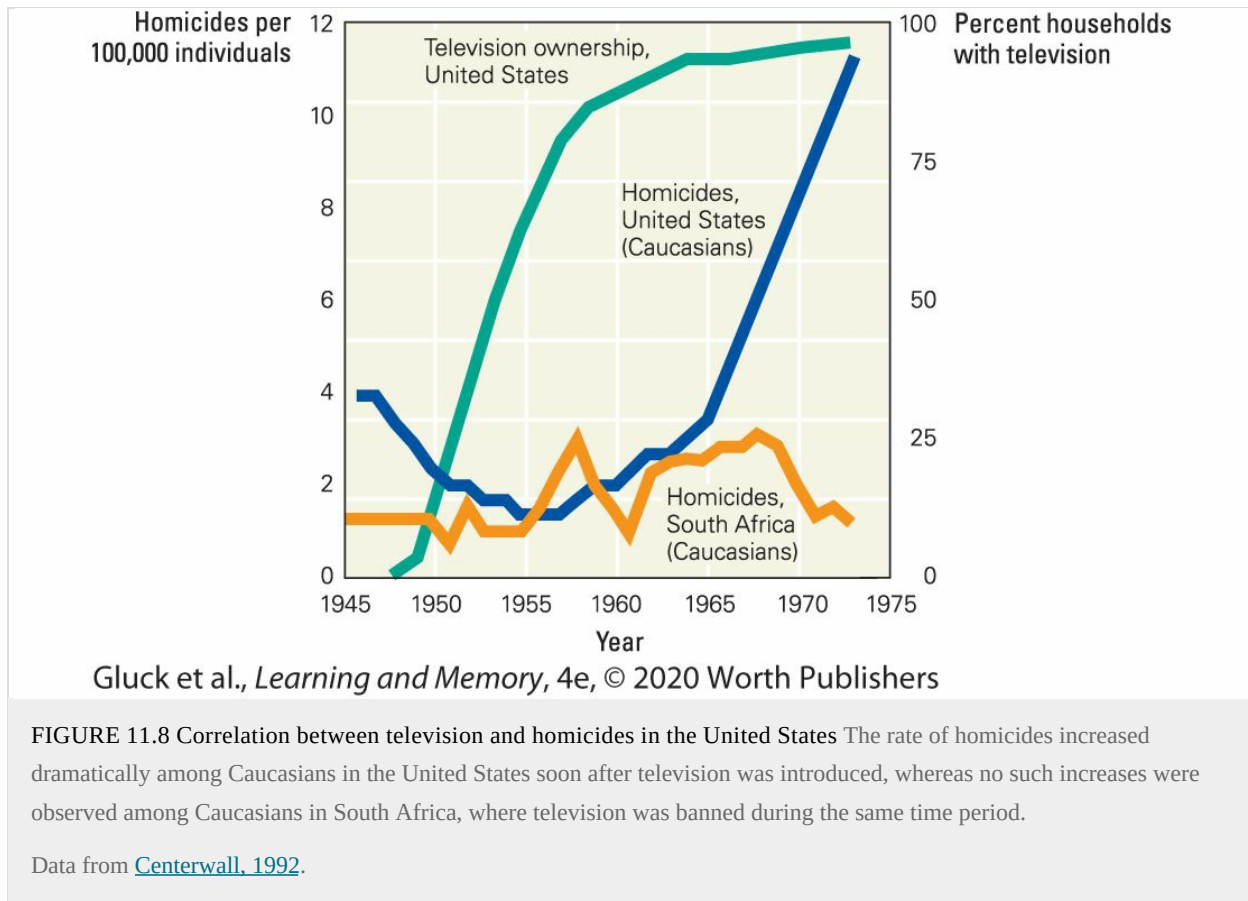


FIGURE 11.8 Correlation between television and homicides in the United States The rate of homicides increased dramatically among Caucasians in the United States soon after television was introduced, whereas no such increases were observed among Caucasians in South Africa, where television was banned during the same time period.

Data from [Centerwall, 1992](#).

Description

The horizontal axis of the graph has years from 1945 to 1975. The left vertical axis shows the number of homicides per 100,000 individuals ranging from 0 to 12, and the right vertical axis shows the percentage of households with television ranging from 0 to 100.

The number of homicides in the United States (Caucasians) was 4 per 100,000 individuals in 1945, and the number was less than 2 per 100,000 individuals by 1955. It started rising gradually during the late 50s and early 60s and stood at 4 again by 1965. It then took a dramatic increase year on year and stood at about 12 homicides per 100,000 individuals by 1973.

The number of homicides in South Africa (Caucasians) in 1945 was less than 2 per 100,000. It plateaued until 1950 and dropped to 1. Following this, it fluctuated over the years, and the number climbed to 4 per 100,000 in 1957. Later, it drastically reduced to 1 per 100,000 individuals but climbed to touch 4 per 100,000 gradually over the years. However, it dropped during the early 70s and was less than 2 per 100,000 individuals by 1973.

The television ownership percentage in the United States sky-rocketed between 1947 and 1975. It was 1 in 1949, 15 in 1950, 60 in 1955, 75 in 1960. It continued to increase to 90 by 1973. All data are approximate.

Most research attempting to address these questions has focused on the effects of viewing violent images. In the typical experiment, researchers randomly assign young participants to watch either a short violent film or a short nonviolent film. Later, the researchers observe the participants' behavior (as in the Bobo doll experiment described earlier). Usually, these observations are made a few minutes or days after the participants view the images, in contexts where aggression might normally occur. Such experiments show that children exposed to images of violence are more likely to behave aggressively in play sessions immediately after viewing the images ([Cornstock, 1980](#); [Geen, 1990](#)). For example, 7- to 9-year-old boys who scored high on measures of aggressiveness were more likely to physically assault other boys in a hockey game after watching a violent video ([Josephson, 1987](#)).

Interactive media such as video games and the Internet have been the subjects of similar research. Part of this interest stems from the fact that students who have gunned down other students in certain school shootings were avid video game players ([Anderson, 2004](#)). Experiments designed to test the negative effects of violent video games compare aggressive behavior in children who have recently played a violent video game with aggressive behavior in children who recently played a nonviolent video game. As in studies of more passive media, children who played the violent video game generally were more likely to behave aggressively ([Anderson et al., 2010](#); [Irwin & Gross, 1995](#)).

Such correlations do not prove that observing violence or playing violent video games increases violent behavior. The same correlations would occur if more aggressive children were more likely to prefer violent games or if increased social isolation led children to act out both virtually and in real-world confrontations. Consequently, the effects of media on aggression are still intensely debated ([Ferguson & Kilburn, 2010](#)). Although researchers have proposed that social learning underlies the association between exposure to violent media and aggressive behavior ([Anderson et al., 2003](#)), most studies of exposure to violent media do not report instances of emulation or true imitation after exposure. In general, it remains unclear what, if anything, individuals learn from observing media that depicts violent scenes. (For further discussion of possible effects of television viewing on behavior, see “[Learning and Memory in Everyday Life](#)”.)

At the core of all current studies of social learning is the idea that simply becoming aware of the actions, interests, mistakes, or strategies of other individuals can change a person's (or an animal's) likelihood of behaving in certain ways in the future, even before any positive or negative outcomes become associated with those behaviors. But how and when such observations might cause individuals to modify their actions remains a mystery. Experimental studies of social learning in nonhumans have opened up new opportunities for identifying and understanding the neural mechanisms that underlie true imitation, emulation, vocal learning, and the social transmission of information, as described in the next section.

LEARNING AND MEMORY IN EVERYDAY LIFE

Learning What to Like from Super Bowl Ads

Breweries spend billions of dollars each year on producing and distributing commercials and other marketing materials intended to increase the probability that consumers will choose their product when deciding which alcoholic beverage to consume. Many of these advertisements are designed to change people's behavior not by making them more informed about the product but rather by associating the product with attractive situations and rewards. In particular, commercials often provide models of drinking behavior that are designed to encourage true imitation or emulation by viewers ([Anderson, de Bruijn, Angus, Gordon, & Hastings, 2009](#)).

Presumably, companies would not be spending such large sums on advertisements if they were not effective. Ads convert watchers into brand-biased consumers through a variety of learning mechanisms, some of which can change a viewer's preferences without the person even knowing it.

For ethical reasons, controlled experiments that show how long-term exposure to beer commercials affect consumption have not been conducted. Correlational studies generally look for associations between familiarity with the content of commercials and drinking behavior, but they usually do not look at details about brand preferences. Such studies have shown that greater exposure to alcohol advertising is correlated with a greater likelihood of binge drinking in young adults ([Hanewinkel & Sargent, 2009](#)).

By portraying attractive people having fun while drinking, commercials not only promote social conformity in viewers but may also associate positive emotional responses with a particular brand. Repeatedly pairing the image and name of a specific company with scenes of happy people drinking may lead to observational conditioning. Likewise, repeatedly pairing a brand with portrayals of humorous situations may lead to classical conditioning of emotional responses.

Super Bowl ads have become famous as a medium through which companies can influence viewers' preferences on a massive scale. The emotional excitement associated with such annual traditions, and with big sporting events in general, can amplify the memories formed during exposure to the advertisements (as discussed in [Chapter 10](#)). This suggests that the alcoholic preferences of football fans are especially likely to be a result of ad-driven conditioning disguised as entertainment.

- Psychologists use *social learning* as a general term for learning from others. Some researchers believe that learning by actively monitoring the acts of others and later choosing actions based on those observations requires specialized learning mechanisms.
- Copying that involves replicating motor acts is called *true imitation*. Copying that involves reproducing an outcome without replicating specific motor acts is called *emulation*.
- In emotional contagion, the observation of a response reflexively evokes that same response. Stimulus enhancement occurs when an individual's attention is directed toward particular stimuli and their associated consequences.
- The ability either to truly imitate or to emulate depends ultimately on the availability of memories for facts or events.
- Many bird species learn to produce songs, but among mammals, only humans and a few species of whales and dolphins are capable of vocally imitating sounds.
- Social transmission of information provides a way for individuals to take advantage of the lessons learned by others.
- Observing others behaving in specific ways can influence the likelihood that an individual will behave in similar ways.

11.2 Brain Substrates

[Section 11.1](#) discussed how social learning often seems to depend both on memories for facts and events (e.g., memories of actions or of socially transmitted information) and on skill memories (e.g., memories of how to perform an action observed in the past). So you might expect that the neural substrates of social learning would be similar to those involved in forming and processing memories for facts, events, and skills. Because only a few species seem to be capable of certain kinds of social learning, however, something beyond these basic neural systems seems to be necessary.

The fundamental problem faced by any individual attempting to replicate an observed action is how to convert perceived events into the motor commands needed to reproduce the observed actions. One possible explanation for how this might be accomplished is the [direct-matching hypothesis](#), which proposes that memories for actions are stored in specialized cortical regions that map observed actions onto the motor representations of those actions ([Buccino, Binkofski, & Riggio, 2004](#)). The direct-matching hypothesis assumes that visually observing an action automatically activates the neural systems required to perform the action and that memories of the observed action are stored as part of this process. The following section describes response properties of cortical neurons that appear to link perceived actions to performed actions in exactly this way.

direct-matching hypothesis

The proposal that memories for actions are stored in specialized cortical regions that map observed actions onto the motor representations of the actions.

Identifying Mirror Neurons

Neurons that fire the same way when an individual performs an action as they do during observations of that action are called [mirror neurons](#) and were first identified in a monkey's cerebral cortex ([di Pellegrino, Fadiga, Fogassi, Gallese, & Rizzolatti, 1992](#); [Gallese, Fadiga, Fogassi, & Rizzolatti, 1996](#)). The electrophysiological experiments that originally led to the discovery showed that certain neurons were firing strongly, both when the monkey grasped an object and when it observed another monkey (or a person) grasping the object. Thus, mirror neurons provide a link between seeing an action and doing that action.

mirror neuron

A neuron that responds during performance of an action and also during observations of that action.

Some mirror neurons fired most strongly during observation of the *outcome* of an action rather than in response to observations of the action itself ([Gallese et al., 1996](#)). This means that the neurons fired the same way when a monkey was seeing a particular outcome (which could be the result of a variety of actions) as when the monkey achieved that outcome itself. So mirror neurons provide the kinds of neural links necessary for both emulation (reproducing an outcome) and true imitation (replicating an observed action). However, there is currently no direct evidence that mirror neurons contribute to social learning in monkeys. Do you think you might have mirror neurons in your brain that help you in social learning?

Mirror neurons are rarely identified in humans because recording the firing patterns of individual neurons requires brain surgery. However, indirect measures of cortical activity, such as electroencephalographs (EEGs; [Raymaekers, Wiersema, & Roeyers, 2009](#)), transcranial magnetic stimulation ([Gangitano, Mottaghy, & Pascual-Leone, 2001](#)), and cerebral blood flow ([Martineau, Andersson, Barthelemy, Cottier, & Destrieux, 2010](#)), indicate that certain regions of the human cortex likely contain mirror neurons ([Figure 11.9](#)). The basic procedure for such studies in humans generally involves an “observation-only” condition, an “imitation” condition, and an “instructed action” condition. For example, researchers might ask participants to (1) observe an image of someone moving a certain finger, (2) imitate someone moving a certain finger, and (3) move a certain finger in response to a specific command. The areas of the human brain that become active during both the observation and performance of actions are located in cortical regions that overlap somewhat with those examined in monkeys, suggesting that monkeys and humans may use similar circuits to match visual observations to actions.

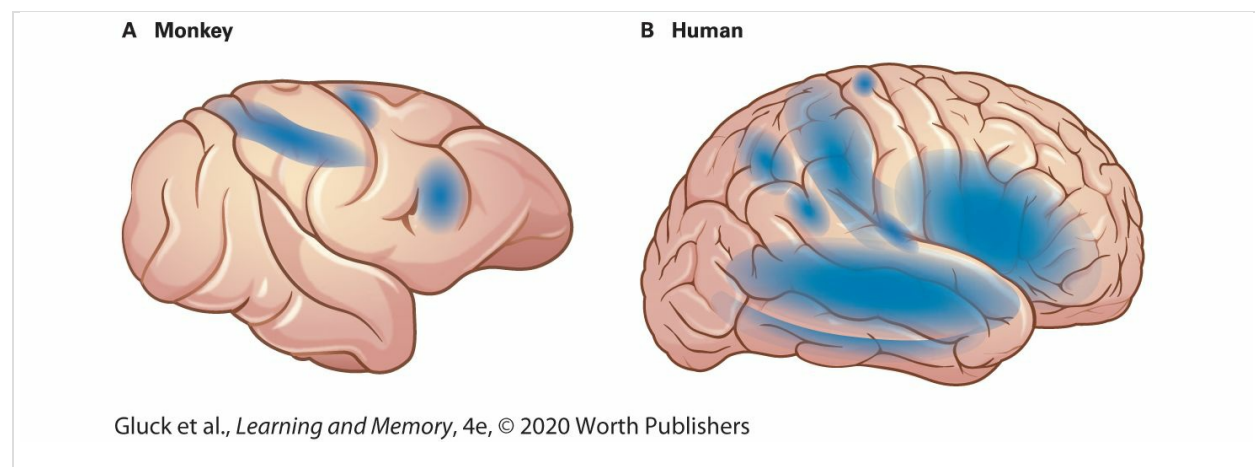


FIGURE 11.9 Mirror neurons in a human cortex? The blue-shaded areas indicate regions in the cortex that became more active in the monkeys (A) or humans (B) when they either watched scenes of others performing an action or performed the action themselves.

Data from [Giret et al., 2017](#).

Description

Illustration A shows the brain of a monkey with small shaded areas near the parietal and frontal regions, and the illustration B shows a human brain with the temporal and frontal regions shaded. These shaded regions correspond to the regions with mirror neurons.

Researchers hypothesize that mirror neurons provide a basic mechanism for true imitation of simple actions ([Rizzolatti & Sinigaglia, 2010](#)). In fact, some researchers suggest that deficits in mirror neurons may contribute to developmental disorders (an idea we discuss in more detail in [Section 11.3](#)). This idea is significant because, in the past, researchers assumed that true imitation involved higher-level cognitive processes that would be difficult to link to specific brain activity in humans and impossible to trace to brain activity in other animals. If mirror neurons are involved in true imitation, emulation, or both, then researchers should be able to examine the mechanisms of imitation in animal models directly.

Mirror neurons have intrigued researchers, in part, because they were an unexpected discovery. As yet, little is known about how they contribute to social learning, although neurostimulation experiments involving transcranial magnetic stimulation (described in [Chapter 2](#)) have shown that disrupting activity in cortical regions thought to contain mirror neurons can disrupt imitation of finger movements ([Catmur, Walsh, & Heyes, 2009](#)). Surprisingly, the most promising advances in understanding the potential role of mirror neurons have come not from studies of imitative primates but from song-learning birds ([Giret, Edeline, & Del Negro, 2017](#)).

TEST YOUR KNOWLEDGE

Who Has Mirror Neurons, and What Do They Do?

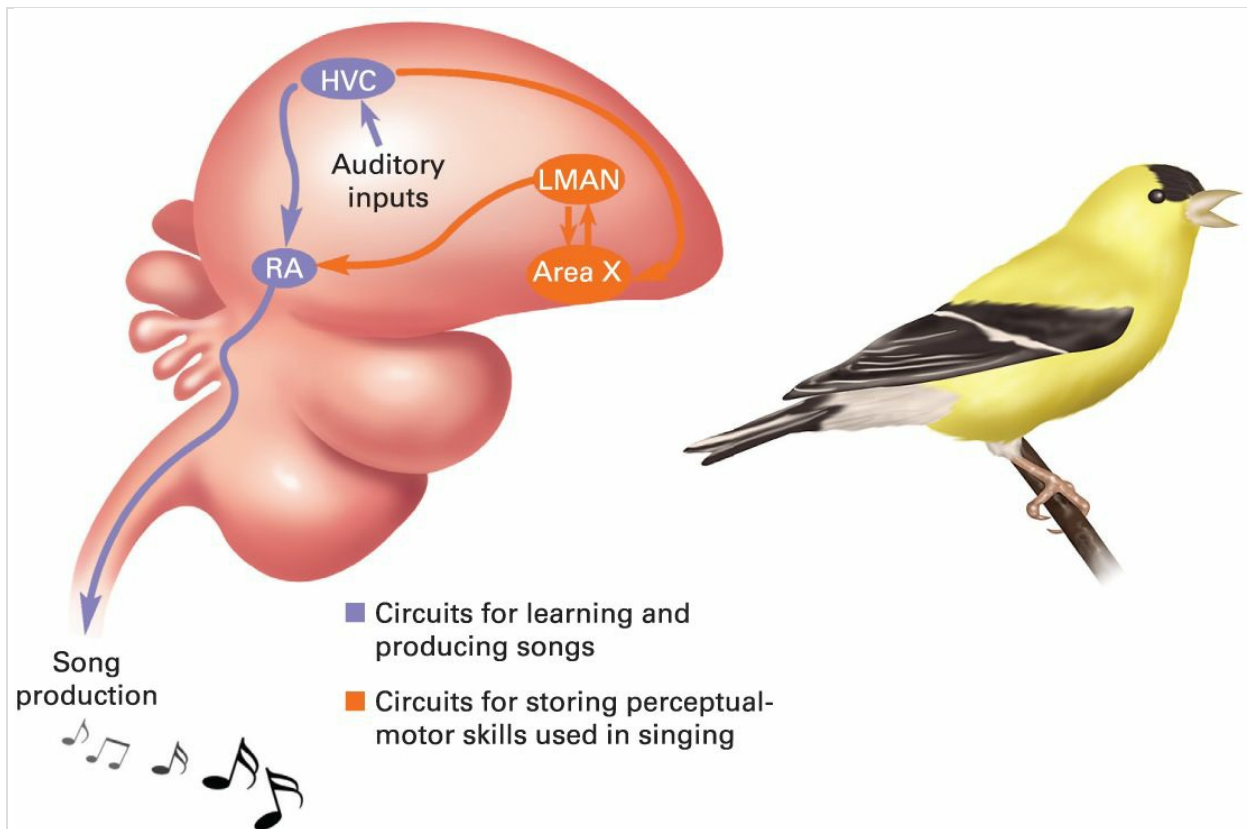
Mirror neurons in monkeys respond the same way to observed actions as to performed actions. On the basis of the preceding discussion, evaluate the following statements about mirror neurons. ([Answers appear at the end of the chapter.](#))

1. Mirror neurons are important for imitative abilities.
2. Mirror neurons in humans respond like those in monkeys.
3. Only primates have mirror neurons.
4. Many mirror neurons in monkeys respond to actions of the hands or mouth.

Mirror Neurons in Songbirds

You might suppose that the neural mechanisms you use for learning from visual observations would be very different from the brain circuits that young birds use when learning to sing. However, these circuits are actually quite similar.

The neural circuits that birds use for learning and producing songs are complex ([Figure 11.10](#)). Birds use specific brain regions to store memories of songs and to learn the perceptual-motor skills for singing. Lesion studies and electrophysiological recordings have identified two main neural regions that birds use for producing songs: the high vocal center (HVC) and the robust nucleus of the archistriatum (RA). The HVC controls the timing of song production—for example, when a bird will start or stop singing and the rate at which notes are produced. The RA controls more detailed features of the individual sounds within a song ([Mooney, 2009](#)). Neurons in the RA that fire strongly when a bird hears a song are also seen to become active just before the bird begins to sing. This means that the RA neurons fire the same way when the bird hears particular songs and when the bird sings those same songs.



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FIGURE 11.10 Neural circuits for song learning by birds Circuits for producing songs and circuits for storing skill memories are shown in different colors. The regions involved in song production include the high vocal center (HVC) and the robust nucleus of the archistriatum (RA). Area X and the lateral magnocellular nucleus of the anterior neostriatum (LMAN) are involved in forming memories. The two circuits are interconnected, permitting memories to affect how songs are produced.

Information from [Brainard & Doupe, 2000](#).

Description

The illustration shows an Eastern Goldfinch bird to the right. The illustration on the left shows the Lateral magnocellular nucleus of the anterior neostriatum (L M A N) sending inputs to Area X and vice versa, which is connected to the high vocal center (H V C) and the robust nucleus of the archistriatum (R A). L M A N is in turn connected to R A. An arrow points to H V C reading, Auditory inputs. The route from H V C to R A and Song production represents the circuits for learning and producing songs. The route from L M A N to Area X and vice versa, L M A N to R A, and the route from Area X to H V C represents the circuits for storing perceptual-motor skills used in singing.

This description should sound familiar to you because it is very similar to the depiction of mirror neuron activity presented earlier. Neurons in the HVC respond both when birds sing and when

they are listening to their own song ([Prather, Peters, Nowicki, & Mooney, 2008](#)). These findings suggest that bird brains contain neurons that map observed events onto vocal acts in a manner comparable with that of the mirror neurons seen in primates. The locations in bird brains where mirror neurons are found also overlap somewhat with analogous locations in primates (compare [Figures 11.9](#) and [11.10](#)).

Birds also possess a neural region called Area X that is thought to be analogous to the mammalian basal ganglia. Lesions to Area X disrupt song learning just as lesions to the basal ganglia of mammals impair perceptual-motor learning (as described in [Chapter 8](#), on skill memories). Area X receives inputs from a region called the lateral magnocellular nucleus of the anterior neostriatum (LMAN). The LMAN is thought to function like the frontal cortex in mammals. Disrupting activity in LMAN neurons when a songbird is first hearing other birds' songs impairs, but does not prevent, song learning ([Basham, Nordeen, & Nordeen, 1996](#)). Similarly, frontal damage in humans can interfere with a person's ability to copy actions, a topic we discuss in greater detail in [Section 11.3](#).

True imitation almost certainly requires a circuit that maps sensory representations onto motor systems, so different species are likely to have developed similar neural solutions to this problem. Future clarification of the neural processes underlying song learning by birds will enable scientists to meaningfully compare the mechanisms underlying social learning of both sounds and sights in the brains of many organisms.

Interim Summary

- In both visually based imitation and vocal imitation, sensory representations are translated into the motor acts necessary to replicate the observed events.
- Mirror neurons fire in the same way when a monkey performs an action as they do when the monkey sees others performing that action, providing a possible mechanism for matching observed actions to motor plans.
- Neuroimaging studies of humans have revealed cortical regions that respond similarly during both the performance and observation of particular actions.
- The song-learning circuits of birds have many features in common with mammalian brain circuits for learning and performing perceptual-motor skills. Mirror neurons are one such feature.

11.3 Clinical Perspectives

Most of the disorders known to affect social learning have an impact on imitative capacities, often in two seemingly paradoxical ways. Patients with imitative deficits cannot seem to prevent themselves from involuntarily copying the actions of individuals with whom they are interacting, yet they have difficulty replicating specific actions when asked to do so. This pattern, which is seen both in some stroke patients and in some individuals with autism spectrum disorder, provides further support for the idea that specific cortical circuits make true imitation possible.

Imitative Deficits After Stroke

Stroke, which is discussed in [Chapter 9](#), is a common source of brain damage. It causes neurons in the affected region to die—in effect, creating a brain lesion. Stroke-related damage in any of several brain regions is known to disrupt imitative abilities, but frontal-lobe damage in particular can have an effect of that kind.

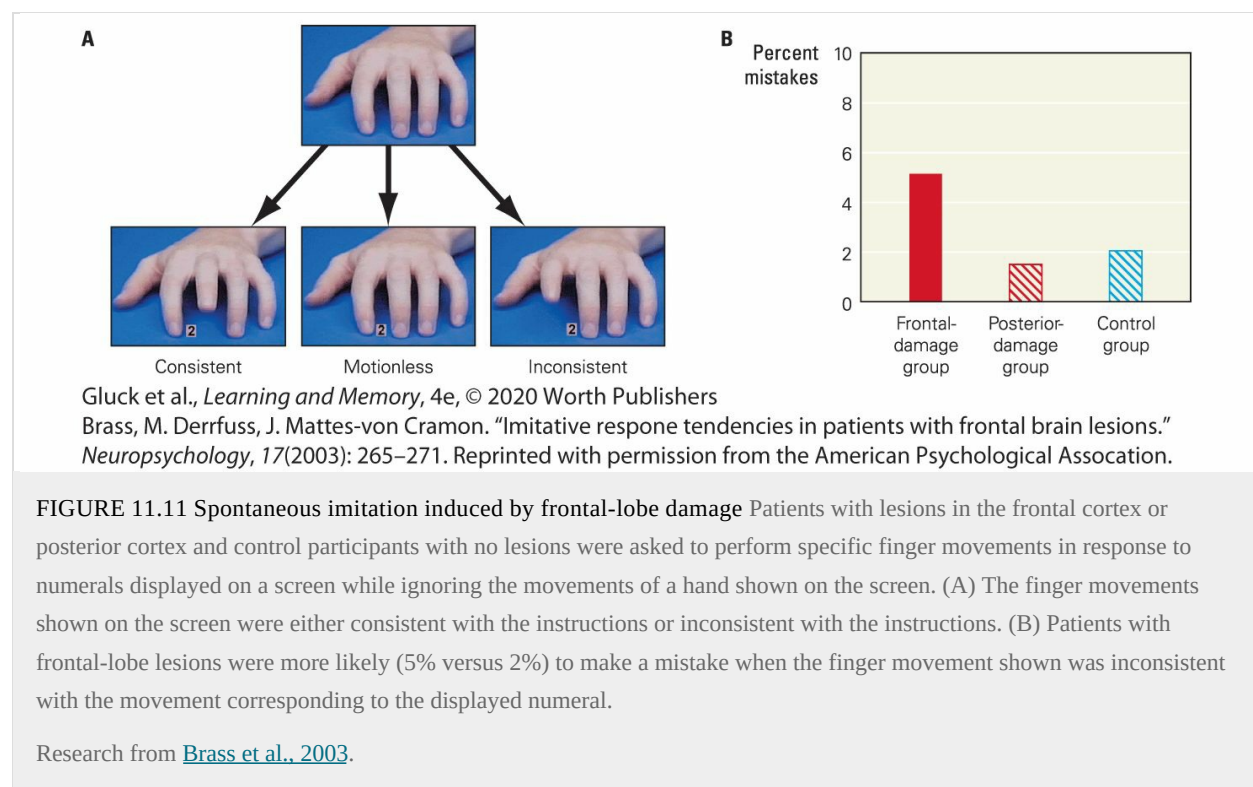
Alexander Luria discovered that patients with frontal-lobe damage tend to produce involuntary imitative responses ([Luria, 1966](#)). This tendency includes [echolalia](#), in which a person repeats words or phrases immediately after hearing them spoken. Luria examined unintentional imitative responses in his patients by instructing them to perform an action at the same time that he performed a related but incompatible action. For example, Luria might show his fist immediately after asking a patient to hold up her palm. Often, seeing Luria make a fist would interfere with a frontal-lobe patient's ability to show her palm when instructed to do so. The patient would tend instead to involuntarily imitate Luria. Patients with reduced activity in their frontal lobes (such as occurs during major depressive episodes) also show this tendency to automatically imitate observed actions ([Archibald, Mateer, & Kerns, 2001](#)). Luria's finding suggests that activity in frontal-lobe circuits normally inhibits this kind of automatic imitation.

echolalia

The automatic repetition of words or phrases immediately after hearing them spoken.

To further explore this tendency, a group of researchers measured the degree to which observations of finger movements interfered with the ability of patients with brain damage to move their fingers correctly in response to commands ([Brass, Derrfuss, Matthes-von Cramon, & von Cramon, 2003](#)). Each patient was asked to lift his or her index finger from a key upon seeing

the numeral 1 and to lift his or her middle finger upon seeing the numeral 2. While performing this task, each patient watched either a video image of a stationary hand, a video image of a hand producing finger movements consistent with the presented numeral, or a video of a hand producing movements inconsistent with the presented numeral ([Figure 11.11A](#)). Frontal-lobe patients made more mistakes than patients with non-frontal lesions when the movement on the video was inconsistent with the instruction ([Figure 11.11B](#)) because they were less able to stop themselves from imitating observed finger movements.



Description

The illustration shows four photos. A photo of a hand resting all the fingers on the table points to three photos below. The first photo labeled consistent shows the middle finger sticking upward while the rest of the fingers resting on the table. The second photo labeled motionless shows no movement of the fingers while they rest on the table. The third photo labeled inconsistent shows the index finger sticking upward while the other fingers rest on the table. All three photos display the number 2.

The graph labeled B shows the three regions of damage on the horizontal axis and percent mistakes on the vertical axis ranging from 0 to 10. The data of the table are as follows.

Frontal damage group, 5.1 percent. Posterior damage group, 1.7 percent. Control group, 2 percent. All data are approximate.

Increases in automatic imitation after a stroke are often correlated with deficits in the ability to voluntarily imitate actions. Note that both conditions suggest a loss of autonomy—specifically, a lost ability to decide or control which observed acts to copy. Cortical circuits in the frontal and parietal lobes may enable people to control whether observations (or memories of observations) will guide their actions, thereby making true imitation and emulation possible.

Autism Spectrum Disorder

Imitative deficits similar to those seen in patients with frontal-lobe damage can also occur in individuals with autism spectrum disorder. **Autism spectrum disorder** is a developmental disorder associated with difficulties in social interactions; a need for highly consistent routines; susceptibility to sensory overload; abnormal language use when communicating with others; and stereotyped movement patterns. For centuries, children who would today be diagnosed as having autism spectrum disorder were grouped with the seriously mentally ill, intellectually disabled, or deaf. Only in the past century have researchers begun to recognize the condition as a disorder having a distinctive pattern of symptoms ([Kanner, 1943](#)).

autism spectrum disorder

A developmental disorder associated with deficits in social interactions and social learning.

Some of the earliest evidence that children with autism spectrum disorder are impaired at copying actions came from the reports of mothers. For example, one mother reported that she was unable to teach her child to play the “patty-cake” game until she physically moved the child’s arms in the correct patterns ([Ritvo & Provence, 1953](#)). If such impairments in copying abilities occur early on in the child’s life, they may possibly contribute to the other behavioral and social deficits of autism ([Rogers & Pennington, 1991](#)).

It remains unclear, however, exactly what social learning abilities might be affected in the disorder ([Beadle-Brown & Whiten, 2004](#); [Hamilton, 2008](#)). People with autism spectrum disorder can recognize when they are being copied, but they do not reliably copy the actions of others ([Leighton, Bird, Charman, & Heyes, 2008](#); [Smith & Bryson, 1994](#); [Vanvuchelen, Schuerbeeck, Roeyers, & De Weerd, 2013](#)). When tested in a do-as-I-do task, children with autism spectrum disorder could copy simple actions, like drinking, but had problems copying sequences of actions ([Beadle-Brown & Whiten, 2004](#)). When tested in the two-action task portrayed in [Figure 11.3](#) (the task with a plastic box containing a food reward), older children

with autism spectrum disorder copied the model's actions normally, but younger children with the disorder did not ([Smith & Bryson, 1998](#); [Whiten & Brown, 1999](#)).

Interestingly, the ability to replicate meaningless gestures (like those used in the patty-cake game) and “nonsense” actions performed with a common object seems more likely to be impaired than does the ability to replicate meaningful actions ([DeMeyer et al., 1972](#); [Smith & Bryson, 1994](#); [Williams, Whiten, & Singh, 2004](#)). At the same time, individuals with autism spectrum disorder often spontaneously (and inappropriately) copy observed speech and actions. It is a bit surprising that individuals with this latter tendency should have difficulty voluntarily imitating meaningless actions. Collectively, the findings described here suggest that there is not an overarching imitative capacity that is “broken” in people with autism spectrum disorder.



Kim Gunkel/Getty Images

Children with autism spectrum disorder often show atypical imitation abilities. Given that adults often rely on demonstration for teaching children to perform tasks, how might these imitation deficits affect what children with autism spectrum disorder learn during their early years?

Researchers have explored whether the mirror neuron system might be contributing to the imitative deficits seen in individuals with autism spectrum disorder ([Ramachandran & Oberman, 2006](#); [Rizzolatti & Fabbri-Destro, 2010](#); [Southgate & Hamilton, 2008](#); [Williams, Whiten, Suddendorf, & Perrett, 2001](#)). Neuroimaging studies show that cortical regions where mirror neurons are thought to be located and patterns of activation within these circuits are atypical in individuals with autism spectrum disorder ([Martineau et al., 2010](#); [Nishitani, Avikainen, & Hari, 2004](#); [Waiter et al., 2004](#)). How such differences affect social learning in those individuals continues to be debated. As our understanding of social learning processes increases, so will our understanding of the roles different brain regions and learning mechanisms play in autism spectrum disorder.

TEST YOUR KNOWLEDGE

Imitative Deficits in Individuals with Autism Spectrum Disorder

Individuals with autism spectrum disorder show several abnormalities in their ability to imitate actions. Which of the statements below would be consistent with imitative deficits observed in these individuals? ([Answers appear at the end of the chapter.](#))

1. A loud sound leads to a panic response.
2. One specific cartoon is repeatedly watched.
3. The individual has problems when trying to keep up with a fitness video.
4. On the phone, the individual often repeats the last phrase she heard.

Interim Summary

- Patients with frontal-lobe lesions tend to copy observed actions automatically but have difficulty copying actions when instructed to do so.
- Individuals with autism spectrum disorder tend to automatically copy others and to have problems copying meaningless actions.

Synthesis

Learning theorists have traditionally assumed that changes in behavior result from the conditioning of passive reflexive reactions (classical conditioning) or from the reinforcement of voluntary responses (operant conditioning). Is social learning anything more than a mixture of classical and operant conditioning? Research into how different brain regions make true imitation and song learning possible is providing new clues. As you might expect, some social learning processes, such as observational conditioning, can be explained as special cases of conditioning. Other abilities—as when humans, apes, and dolphins imitate actions on command—are more difficult to account for in terms of incremental-learning models and instead seem to require unique cortical-processing mechanisms.

Understanding what happens when imitative mechanisms go awry can also lead to new ideas about how social learning works. Correlating the imitative deficits in individuals with autism spectrum disorder with those of stroke patients may provide insights into the relationship between automatic and voluntary copying. The fact that in both conditions echolalia tends to co-occur with involuntary imitation of actions suggests that vocal and visually based imitation may rely on overlapping neural systems.



moodboard/Corbis

What sorts of social learning are professors relying on when they give lectures?

Albert Bandura has suggested that social learning is a process that permits rapid, errorless acquisition of cognitive and perceptual-motor skills and that humans who take full advantage of this process have “learned to learn.” This acquisition of knowledge through social transmission of information is often described as being unique to humans and is closely tied to language abilities. You are potentially engaging in such observational learning at this very moment. Social transmission of information through lectures and textbooks forms the basis for learning in most courses. More modern perspectives on social learning suggest that humans are not the only animals that use past observations to guide their choices of actions and that studies of other species may be critical to identifying how social learning mechanisms are related to other learning and memory mechanisms. Perhaps educated dolphins like Akeakamai can one day help researchers discover the basic principles of learning from others’ mistakes and successes.

Chapter 11 Review

Know Your Key Terms

[autism spectrum disorder](#)

[copying](#)

[direct-matching hypothesis](#)

[echolalia](#)

[emotional contagion](#)

[emulation](#)

[mirror neuron](#)

[modeling](#)

[observational conditioning](#)

[perspective taking](#)

[social conformity](#)

[social learning](#)

[social learning theory](#)

[social transmission of information](#)

[stimulus enhancement](#)

[template model of song learning](#)

[true imitation](#)

[two-action test](#)

[vocal imitation](#)

[vocal learning](#)

Quiz Yourself

1. A child who wildly beats an inflatable doll after seeing adults do so provides evidence of _____ learning because his observations have produced a change in his behavior. (p. 468)
2. A toddler who makes a puppet dance after observing her sibling do so may be _____ her sibling. (p. 469)

3. A chimpanzee that learns to open doors by banging them with its feet after seeing humans open doors with their hands is likely showing evidence of _____. (p. 472)
4. The two-action test requires training _____ to serve as models performing a task that observers will watch. (p. 472)
5. Researchers have used the two-action test to determine whether animals are capable of _____. (p. 472)
6. The ability to imagine what it would be like to appear in an advertisement requires _____. (p. 474)
7. The reason that laugh tracks are used so often in sitcoms is that hearing laughter leads to _____. (p. 475)
8. If crows learn to attack all clowns after seeing other crows attacking clowns, then this is likely a case of _____. (p. 476)
9. In observational conditioning, observation of a model produces a(n) _____. (p. 476)
10. If one crying baby on an airplane triggers a flurry of crying infants, then this is likely due to _____. (p. 475)
11. A model on a game show waving her arms in front of a prize that contestants might win is an example of _____. (p. 477)
12. True imitation differs from _____ in that true imitation requires replicating the actions of a model. (p. 472)
13. Stimulus enhancement may be mistaken for _____ if the actions of the observer match the actions of the “model.” (p. 477)
14. Birds are important subjects in studies of neural and behavioral social learning because of their _____ ability. (p. 481)
15. One form of imitation that is rare among mammals is _____. (p. 479)

16. A(n) _____ is an example of an animal that can imitate both sounds and actions. ([p. 479](#))
17. Speech imitation in humans is an example of _____. ([p. 479](#))
18. The fact that some songbirds raised alone never sing normally is evidence that _____ is important for their song development. ([p. 481](#))
19. The _____ suggests that songbirds memorize the songs of adults before learning to sing them. ([p. 482](#))
20. An observer rat learns to prefer a certain food from exposure to other rats. This is an example of _____. ([pp. 484–485](#))
21. If you take notes in class because other students do, but you never actually look at them, you could be demonstrating _____. ([p. 485](#))
22. The _____ states that observations are directly mapped onto motor representations. ([p. 489](#))
23. Cells that respond when a monkey grabs a stick and also when the monkey sees someone grab a stick are called _____. ([p. 489](#))
24. Brain regions in humans that respond similarly when a person performs finger movements or observes finger movements are found in the _____. ([pp. 489–490](#))
25. One of the brain regions active in avian song learning is _____. ([p. 491](#))
26. Children with autism spectrum disorder often repeat sounds or sentences they have recently heard, a phenomenon known as _____. ([p. 492](#))
27. Because individuals with autism have difficulty performing certain imitation tasks, it has been theorized that they have dysfunctional _____. ([p. 494](#))
28. Patients with _____ show a tendency to imitate actions involuntarily. ([p. 492](#))
29. Findings from Bandura's _____ study have been used to argue that children

exposed to violence are more likely to behave violently. ([pp. 470, 486](#))

Concept Check

1. Some researchers have suggested that the only way to be sure that you are observing imitation is if the actions or sounds being copied are so bizarre that there is no other plausible explanation for why they might occur ([Thorpe, 1963](#)). What are some other ways researchers can tell when an animal is truly imitating?
2. [Edward Thorndike \(1898\)](#) defined imitation as learning to do an act from seeing it done, but visual observations are not the only way to get the information needed to imitate or emulate. What are some examples of copying described in this chapter that would not count as imitation under Thorndike's definition?
3. In the wild, animals may gain an advantage from spending more effort investigating areas that have been occupied by other members of the same species. Such behavior could increase the chances of finding food, mates, or a safe place to hide. What forms of social learning might lead animals to explore such areas?
4. Children may (a) be born with an ability to copy that depends on specialized mechanisms for matching visual perceptions to motor responses ([Kuhl & Meltzoff, 1996](#); [Piaget, 1955](#)), (b) learn to copy through experience, and (c) consciously consider the perspective or social status of other individuals when they copy ([Guillaume, 1971](#)). For which of these types of learning have neural substrates been identified?
5. [Albert Bandura \(1986\)](#) argued that operant conditioning without imitation is an inefficient way of learning new actions and that only humans learn by copying because learning from models requires abstracting rules and forming memories based on verbal codes and imagery. List information from this chapter that either supports or refutes these ideas.

Answers to Test Your Knowledge

Is It True Imitation?

1. Perhaps the baby is imitating you. However, another possibility is that smiling faces are a reinforcing stimulus for the baby. Reinforced babies are more likely to smile, and thus the baby may be responding to you in the same way that she would if you had handed her a cookie (another reinforcing stimulus). No copying is required.
2. This could just be a case of contagion. If a few pigeons in a group start to fly, there is a good chance that you will see other pigeons start to fly—not because they are copying each other but as a reflexive response (the same as when schooling fish move in synchrony). To assess this possibility, you need to know more about what causes babies to stick out their tongues.
3. This could be a case of observational conditioning. Perhaps Iam has seen his father eating asparagus several times. Seeing his father express disgust might induce similar negative emotions in Iam. The asparagus could become a conditioned stimulus that leads to a conditioned response of disgust. Alternatively, maybe this is the first time Iam has tried asparagus, and he simply does not like it.
4. Darwin thought so. However, the cat might have been trained to perform these actions through shaping. (Such training of waterskiing squirrels is described in [Chapter 5](#), on operant conditioning.) Alternatively, this could be a case of stimulus enhancement. Humans approaching the door always focus their actions on the doorknob, which might direct the cat's attention to the doorknob as well. Cats often hit the objects of their attention with their paws. A few instances of accidental door opening would likely increase the cat's tendency to swat at the doorknob.
5. Yes. Iam's friend is replicating Iam's actions in a way that cannot be explained as anything other than imitation (unless they are both reading from scripts).

Who Has Mirror Neurons, and What Do They Do?

1. There is no direct evidence in monkeys or humans that mirror neurons are necessary for imitative abilities, so whether they are important or not is not yet known.
2. There are currently no measures of recordings from mirror neurons in humans that are

directly comparable to those from monkeys, so this is currently unknown.

3. Primates are the only species in which mirror neurons related to visual observations of actions have been observed, but this does not provide evidence that other animals do not also have mirror neurons.
4. This is true.

Imitative Deficits in Individuals with Autism Spectrum Disorder

1. This is a symptom seen in some individuals with autism spectrum disorder (ASD), but it has nothing to do with imitation or social learning.
2. Narrow interests are a symptom of ASD but do not depend on imitative abilities.
3. Fitness videos often require viewers to imitate arbitrary actions, so this difficulty could reflect an imitative deficit.
4. This is an example of echolalia, which is an imitative deficit often seen in individuals with ASD.



Chapter 12 Development and Aging

Learning and Memory Across the Lifespan



By Kurt Stricker/Getty Images

Behavioral Processes

[The Developing Memory: Infancy Through Childhood](#)

[Sensitive Periods for Early Learning](#)

Learning and Memory in Everyday Life: Teaching Babies Signs Before Speech

Adolescence: Crossing from Childhood into Adulthood

The Aging Memory: Adulthood Through Old Age

Brain Substrates

The Genetic Blueprint Is Modified by Experience

Learning and Memory in Everyday Life: Can Exposure to Classical Music Make Babies Smarter?

Neurons and Synapses in the Developing Brain

Brain Changes in Adolescence

The Brain from Adulthood to Old Age

Clinical Perspectives

Down Syndrome

Alzheimer's Disease

A Connection Between Down Syndrome and Alzheimer's Disease

Learning and Memory in Everyday Life: Can Mental Exercise Protect Against Alzheimer's Disease?

TWENTY-YEAR-OLD DENISE IS ENTERING THE PRIME OF HER LIFE. As a state champion on the basketball court, her body is at the peak of its physical strength and stamina. As a college student, she challenges her brain every day to learn new skills, organize and retrieve information for exams, and integrate new knowledge—including what she studies in her learning and memory course—into her broadening understanding of the adult world.

At Thanksgiving, Denise goes home to visit her family and plays with her young nieces and nephews. The youngest, Kelly, is a 10-month-old, busily soaking up information about the world. Kelly is just learning to stand and walk, to recognize and respond to spoken words, and to produce babbling sounds that will someday lead to fluent language.

Among the oldest family members gathered for the holiday is Denise's grandmother: a wise, funny woman, and a walking library of family history and special recipes. But Denise's grandfather has gone downhill since last Thanksgiving. He repeats the same stories over and over again and has trouble remembering Kelly's name. The family worries that this is not just normal aging but a sign of something more serious, such as Alzheimer's disease. The specter of this worry hangs over the holiday celebration.

This chapter traces the development of learning and memory abilities across the lifespan, starting with infants like Kelly and culminating with elderly individuals like Denise's grandparents. You'll recognize many types of memory that were introduced in earlier chapters, but here we'll view them from the standpoint of how a person's memory changes over the lifespan and how this reflects underlying age-related changes in the brain. You'll also encounter topics that may be new to you, such as the emerging field of epigenetics, the effects of sex hormones on developing and mature memories, and the concept of sensitive periods—"windows of opportunity"—for specific kinds of learning, after which such learning may never again be so easy or effective.

12.1 Behavioral Processes

One reason to study learning and memory across the lifespan is to appreciate the different capabilities of children, teenagers, mature adults, and older adults. Understanding which types of learning and memory are comparatively strong or weak at each age can help us all make the most of our potential at every stage of life. It can also help educators tailor their teaching methods to different age groups.

As you read, though, be sure to keep in mind that we are talking about the *average* learning and memory abilities of groups at various ages. Within any age group, there is wide variation in the abilities of individuals. Both genetics and environment play a role, so some youngsters will inevitably demonstrate a different pattern of development, acquiring new abilities more quickly or more slowly than their peers. Similarly, at the other end of the lifespan, some individuals retain relatively strong learning and memory abilities even into extreme old age, while others lose cognitive abilities at an accelerated rate due to injury or illness such as Alzheimer's disease.

With these thoughts in mind, let's begin at the beginning: with the learning and memory of individuals at the earliest stages of life.

The Developing Memory: Infancy Through Childhood

If you have spent any time with a young child, you have witnessed the immense surge of learning that takes place in the first few years of life. In those years, a child acquires a broad range of abilities, including the motor skills needed to grasp and walk, a basic competency at producing and understanding language, and a capacity for compiling semantic knowledge about the world. Learning continues across the lifespan, of course, but its progress is especially dramatic in young children. More recently, it has become apparent that some learning occurs even before birth.

Learning Before Birth

By about 6 weeks of **gestational age**, or time since conception, the fetus starts making simple, stereotyped movements that allow it to feel the space around it and sense the action of its body ([Fagard, Esseily, Jacquey, O'Regan, & Somogyi, 2018](#)). For the next few weeks, the fetus

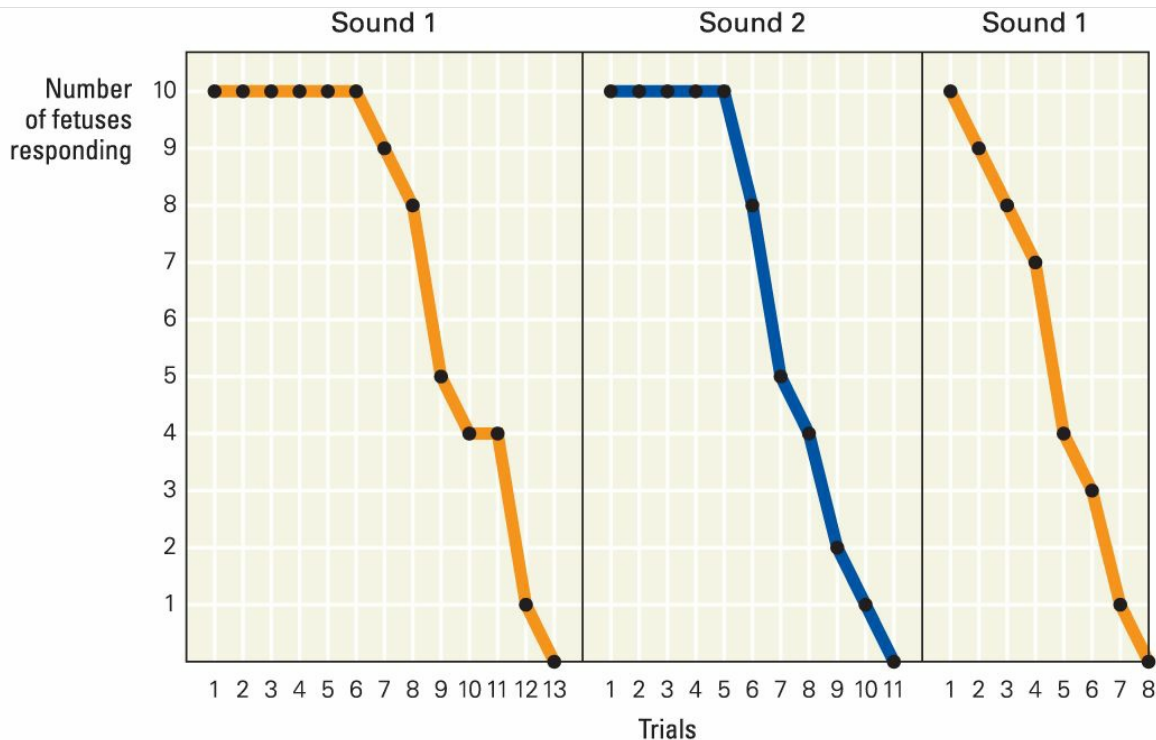
explores its environment and its body, discovering contingencies; for example, when the brain issues a certain motor command, a limb moves, resulting in a consequence, such as the sensation of pressure as the arm pushes against the wall of the uterus. This early “motor babbling” helps organize the motor cortex, which will eventually develop specialized subregions to control different body parts (the “motor homunculus” shown in [Figure 2.14](#)).

gestational age

Time since conception.

Meanwhile, the brain is also learning to process and respond to sensory stimuli. A human fetus is exposed to the sounds of maternal speech and heartbeats as well as noises from the outside world. By about 25 weeks of gestational age, the brain and sense organs are sufficiently developed for the fetus to start perceiving and learning about these sounds.

For instance, [Chapter 3](#) describes *habituation* as the phenomenon of reduced responding to a repeated stimulus. Habituation can be used to test fetal learning about sounds. Researchers place a speaker against the mother’s abdomen and play sounds that the fetus can hear. The first time a sound plays, most human fetuses with gestational ages of 34 to 36 weeks will respond by moving ([Figure 12.1](#)). If the same sound is played several times, the fetuses gradually stop responding ([Hepper & Shahidullah, 1992](#)). When a different sound is played, the responses reappear—indicating that the loss of response to the first stimulus was due to habituation, not fatigue. Eventually, responses to the second stimulus habituate, too. When the first sound is played again, the fetuses respond but habituate almost immediately, indicating that they “remember” having experienced that sound before.



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

FIGURE 12.1 Habituation to sound in 10 human fetuses When sound 1 is presented, all 10 human fetuses initially respond with movement; as the sound is repeated, the number of fetuses responding decreases. By trial 13, all have habituated. When a new stimulus (sound 2) is presented, all the fetuses again respond; this response also habituates over about a dozen trials. Finally, the original stimulus (sound 1) is presented again. The fetuses show spontaneous recovery of responding followed by fast habituation (only 8 trials).

Information from [Hepper & Shahidullah, 1992](#).

Description

The graph has trials from 1 to 13 on the horizontal axis and number of fetuses from 1 to 10 on the vertical axis.

Sound 1: all the 10 fetuses respond to sound 1 for 7 trials after which the number drops significantly from 9 to 5 fetuses up until the eighth trial. The number further reduces to 4 fetuses by the tenth and the eleventh trial. No fetus responds to sound 1 by the thirteenth trial.

Sound 2: all the 10 fetuses respond to sound 2 for 6 trials after which it drops to 8 and further to 5 fetuses by the fifth trial. The number of fetuses responding to sound 2 continues to be fewer as the trials increase and by the eleventh trial no fetus shows response.

Sound 1: all the 10 fetuses respond to sound 1 again during the first trial and the number reduces sooner with each subsequent trial indicating faster habituation. By the eighth trial no fetus shows response.

In addition to recording movements, researchers can record fetal brain responses, using *fetal*

magnetoencephalography (*fMEG*), a form of functional imaging in which sensors placed against the mother's abdomen measure the magnetic fields generated by neuronal activity in the fetus's brain ([Sheridan, Matuz, Draganova, Eswaran, & Preissl, 2010](#)). *fMEG* studies show high brain activity in fetuses the first few times an auditory stimulus is played. Just like the motor behavior shown in [Figure 12.1](#), the brain activity habituates with repeated presentation of the same tone—but rebounds when a new tone is played ([Muenssinger et al., 2013](#)). Fetal habituation can be observed in nonhuman species, too, including rats ([Smotherman & Robinson, 1992](#)).

Such prenatal learning can persist after birth. Researchers Anthony DeCasper and Melanie Spence asked a group of pregnant women to participate in a study. They gave each woman a short story with a strong rhyming pattern, such as an excerpt from Dr. Seuss's *The Cat in the Hat*, and asked each mother to read that story aloud to her unborn baby twice daily during the last 6 weeks of pregnancy ([DeCasper & Spence, 1986](#)). Would the infants later recognize the familiar sounds?

Beginning 2 or 3 days after birth, each newborn was placed in a crib with an artificial nipple positioned where the infant could suck at it. Under these conditions, infants usually suck in bursts: giving a series of brisk individual sucks, pausing a few seconds, and then giving another burst of sucks. For this experiment, the nipple was rigged so that each burst of sucking caused a tape recorder to turn on and play a story recorded in the mother's voice. For one set of babies, if a sucking burst followed a longer-than-average pause, then the familiar story played; if a sucking burst followed a shorter-than-average pause, then an unfamiliar story played:

S^D (artificial nipple) → R (long pause before sucking burst) → O (familiar story)

S^D (artificial nipple) → R (short pause before sucking burst) → O (novel story)

Over a period of 20 minutes, the infants changed their sucking behavior, timing their pauses in such a way as to result in the familiar story being played more often. In short, the familiar story reinforced a particular pattern of sucking behavior. This learning is an example of *operant conditioning*, as discussed in [Chapter 5](#). Obviously, the babies did not understand the meaning of the words in either story; the difference in their responses probably reflected an ability to discriminate the cadence and rhythm of the two stories. Still, the study demonstrates that prenatal

infants are capable of learning and that the learned information persists and can affect behavior after birth. DeCasper and colleagues suggest that fetal exposure to the mother's language patterns may help the brain start to encode language-relevant speech sounds, giving the babies a head start on acquiring language after birth ([DeCasper, Lecanuet, Busnel, Granier-Deferre, & Maugeais, 1994](#)). Studies such as this show that newborns come into the world not as blank slates but having already experienced stimuli and begun to learn about them.

Conditioning and Skill Learning in Young Children

Despite evidence for fetal learning, the really impressive learning machines are infants. An explosion in learning marks the first few years of life in humans and analogous periods in other species. Just about every kind of learning that is present in adults is present in infants—at least in a rudimentary form. On the other hand, some of a newborn's perceptual and motor systems are still immature. For example, cats are born with their eyes sealed shut, so they obviously cannot learn much about visual stimuli that are presented to them in the first few hours of life. Human newborns have visual systems that can detect changes in brightness and movement, but their visual acuity is weak. Acuity improves over the first 2 months of life, allowing infants to perceive fine detail and, eventually, identify particular patterns and objects. By 2 weeks after birth, newborns can recognize their mothers' faces, as indicated by longer looking at photos of their own mother than at photos of strangers ([Bushnell, 2001](#)).

For humans, acquisition of *complex motor skills* comes gradually, as physical development produces improvements in muscle strength and perceptual–motor coordination. Newborn humans cannot even hold their heads up unsupported, but most babies can roll over by the time they are 5½ months old; by 7 months, most can sit up; by 1 year, most have learned to stand alone or even walk. As coordination improves, babies develop the fine motor skills needed for accurate eye tracking, babbling, and reaching to pick up objects. Skill acquisition is not limited to motor skills either; between 1 and 2 years of age, children begin to master the rudiments of language, and complex grammar and reading are usually evident by 4 to 5 years.

You read earlier that infants can show operant conditioning, learning particular sucking responses that produce outcomes such as tape recordings turning on and off. In another operant conditioning procedure, researchers hang a mobile over an infant's crib and tie one end of a ribbon to the baby's leg and the other end to the mobile. When the baby kicks her leg, the mobile

moves:

S^D (crib with mobile) → R (kick) → O (mobile moves)

Just like a rat in a Skinner box, the baby makes her first response by chance. The resulting movement of the mobile, which babies find highly entertaining, reinforces this kicking response. Babies as young as 2 months of age quickly learn to kick vigorously to produce mobile movement ([Rovee-Collier, 1993](#), [1997](#), [1999](#)). The babies can maintain memory of this learned response for a few days with no reminders—or for up to 21 weeks if they receive periodic reminders in which they see the mobile move when an out-of-sight experimenter pulls the ribbon ([Hayne, 1996](#)).



In memory of Carolyn Rovee-Collier

This infant has learned through operant conditioning that when she kicks her leg, the mobile moves. After the infant has learned this association, what do you think would happen if the ribbon connecting the infant's ankle to the mobile were removed?

[Chapter 6](#) discusses how learning is often context dependent. Operant learning in babies is an example. In one variant of the mobile experiment, learning takes place in a crib with a striped crib liner. If this liner is then replaced with one having a different pattern, the babies gape passively at the mobile but do not kick ([Borovsky & Rovee-Collier, 1990](#)). Apparently, the

change in crib liners represents a new context in which the old rules might not apply. Infants, just like adults, incorporate details of the context during ongoing learning.

Classical conditioning also takes place in infants. [Chapter 4](#) describes eyeblink conditioning, in which a human or a rabbit learns that a tone or light (the conditioned stimulus, or CS) predicts an airpuff (the unconditioned stimulus, or US). In the *delay-conditioning* paradigm, the CS and US overlap and co-terminate. With repeated CS–US pairings, the human or rabbit learns to respond to the CS by producing an eyeblink (the conditioned response, or CR) so that the eye is protected when the US arrives (see [Figure 4.8](#)).

Infant humans can learn eyeblink CRs in the delay-conditioning paradigm, and so can infant rats, although in both species the infants learn more slowly than adults do ([Ivkovich, Collins, Eckerman, Krasnegor, & Stanton, 1999](#); [Ivkovich, Paczkowski, & Stanton, 2000](#); [Little, Lipsett, & Rovee-Collier, 1984](#)). By contrast, in the more difficult *trace-conditioning* paradigm, where there is a gap between the end of the CS and the arrival of the US, 2-month-old infant humans cannot learn the eyeblink CR at all. Four-year-olds can learn in the trace-conditioning paradigm, but they do so more slowly than they learn in the delay-conditioning paradigm; in contrast, young adult humans learn both paradigms about equally quickly ([Ivkovich et al., 1999](#); [Ivkovich et al., 2000](#); [Little et al., 1984](#)). In summary, the basic components of classical conditioning are available in very young mammals, but the capacity continues to develop as the individual matures, allowing learning to proceed more efficiently and under increasingly difficult conditions.

An important aspect of conditioning (and all other learning) is *generalization*, the ability to transfer past learning to new situations (as explored in [Chapter 6](#)). One way to assess generalization is through tests of *acquired equivalence*, in which learning about one stimulus generalizes to other stimuli that have been equivalent in the past (see [Figure 6.21](#)). For example, a child raised in a bilingual home may observe her English-speaking grandparents using one set of labels to refer to objects (“cat,” “bottle”), while her Spanish-speaking grandparents use different labels for the same objects (“gato,” “botella”). If she hears her Spanish-speaking grandmother refer to a new object as “perro,” she will infer that her other Spanish-speaking relatives, but not the English-speaking ones, will use that same word. In the laboratory, 8-month-old infants show surprise when such rules are violated, indicating that this form of generalization

is present even at a very young age ([Werchan, Collins, Frank, & Amso, 2015](#)).

In fact, accumulating evidence suggests that infants are active learners who develop sophisticated hypotheses about how the environment behaves, use generalization to make predictions based on these hypotheses, and notice (and react with surprise) when these predictions are violated ([Xu & Kushnir, 2013](#)).

Development of Episodic Memory and Semantic Memory

As discussed in [Chapter 7](#), *semantic memory* involves factual knowledge, whereas *episodic memory* is memory for specific autobiographical events, including the spatial and temporal contexts in which the events occurred. Both episodic memory and semantic memory are most often tested in adult humans by asking participants whether they can recall (or recognize) studied material, but this is obviously not possible with preverbal infants. So researchers have devised creative techniques that allow infants to express their knowledge, even if they can't yet speak about it.

One technique for assessing memories in preverbal infants is [elicited imitation](#), in which infants are shown an action and tested for their ability to mimic this action later ([Bauer, 1996](#)). For instance, researchers showed a group of 10-month-old children how to operate a toy puppet. Four months later, they presented the children with the same toy puppet. These children showed more interest and ability in operating the puppet than did a second group of same-age children who had never seen the puppet used ([Myers, Perris, & Speaker, 1994](#)). This difference suggests that the very young children had at least some memory of the puppet, even if they couldn't verbalize it ([Rovee-Collier, 1999](#)).

elicited imitation

A technique for assessing memory in infants by observing their ability to mimic actions they have seen earlier.

Once children grow a little older and start to acquire language, it becomes much easier to investigate semantic memory. During the first few months of age, children begin to imitate the speech sounds they hear around them; and by about 1 to 2 years of age, babies learn and use individual words, usually acquiring a vocabulary of several hundred words (mostly nouns and verbs). Over the next few years, vocabulary continues to develop, and children also begin to

acquire a knowledge of *syntax*, or the rules governing how words are arranged into sentences. In addition, children learn about physical objects and events; a toddler's incessant "Why?" questions reflect how she is soaking up semantic information about the world. Much of this information, acquired in early childhood, remains with us for life.

Episodic memory matures more slowly than semantic memory. One factor may be that parts of the brain, such as the hippocampus and prefrontal cortex, which are critical for encoding and recall of episodic memories, are immature in humans at birth and continue to develop throughout childhood ([Yim, Dennis, & Sloutsky, 2013](#)). Another factor may be that very young children do not show evidence of a "cognitive self." One widely used test of the sense of self is whether an individual can recognize herself in the mirror. In one test, a researcher surreptitiously marks the individual's face with rouge; if the individual views herself in the mirror and touches the marked spot on her face (as if to investigate or wipe off the mark), then we may conclude that she recognizes the image as her own ("Hey, that's me—and what's that red dot doing on my nose?"). Adult humans, as well as adults of a few species such as chimpanzees, bottlenose dolphins, and magpies, all respond to mirrors with behavior that seems to indicate they recognize their own images (for review, see [de Waal, 2019](#)).



Angela Georges/Getty Images

As a test of self-awareness, a red dot is placed on a child's face, and the child is placed in front of the mirror. If the child tries

to wipe the dot off her actual face, researchers conclude that she recognizes her own reflection, which is often taken as evidence of self-awareness and a sense of self. Why might a sense of self be a prerequisite for episodic memory?

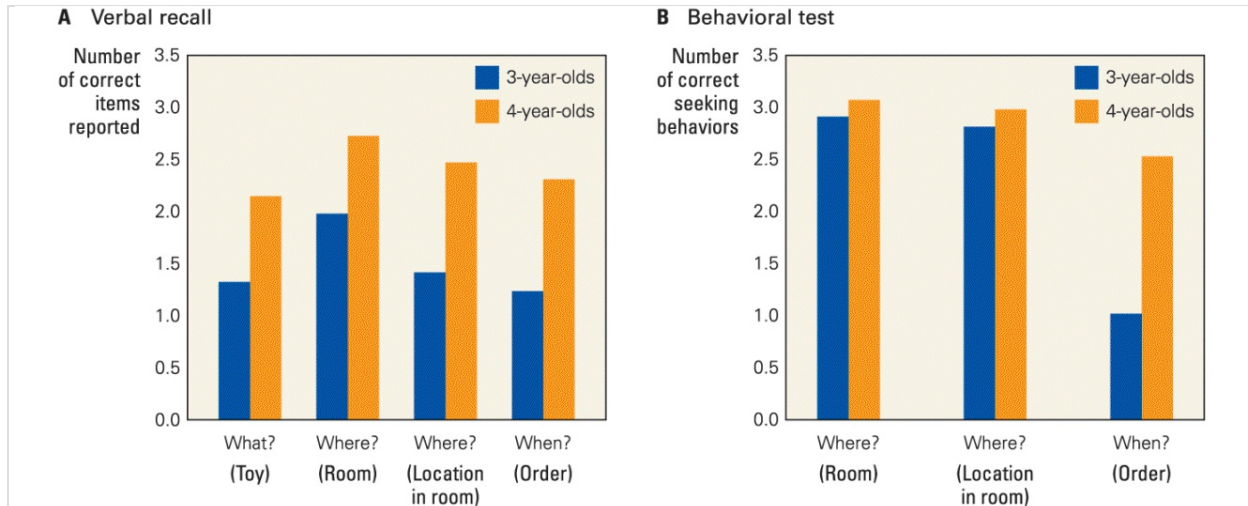
Infants younger than 16 months do not show mirror-recognition behavior, but children older than 24 months do ([Lewis & Brooks-Gunn, 1979](#)). This implies that 2-year-olds, but not 1-year-olds, have a sense of themselves as individuals. This cognitive milestone is probably a prerequisite for forming autobiographical memories ([Howe & Courage, 1993](#)). You can't remember that a particular event happened to you unless you have a sense of yourself and how you exist in time ([Fivush & Nelson, 2004](#)).

On the other hand, some infancy researchers argue that even very young children can and do form episodic memories; they just may not be able to communicate them verbally the way older children can ([Rovee-Collier & Cuevas, 2009](#)). If so, the “failure” of young children to display episodic memory may really be a failure of the researchers' experimental design.

[Chapter 7](#) describes an experiment in which researchers assessed “episodic-like” memory by observing scrub jays digging for worms and nuts in an ice cube tray ([Figure 7.3](#)). The birds displayed evidence of remembering what, where, and when the items were buried, suggesting that the birds had a memory for the specific episode, including the spatial and temporal context, in which the burying occurred. Other researchers developed an analogous task for young children based on the game of hide-and-seek ([Hayne & Imuta, 2011](#)). Children may not be motivated to dig for worms and nuts, but they do like toys and are highly motivated to retrieve them. In the task, children hid three different stuffed toys in three separate locations around their own homes. Later, experimenters asked the children to verbally recall what was hidden (e.g., “the Eeyore toy”), where (“the bedroom”), and when (“the last time we entered”).

Unsurprisingly, 4-year-old children provided more correct answers than 3-year-old children did, as shown in [Figure 12.2A](#). But then the experimenters gave a behavioral test, asking the children questions such as, “Can you show me the room we went into first and find who is hiding there?” Now the 3-year-olds were just as good as the 4-year-olds at remembering what was hidden where, although they were still not quite as good as the older children at remembering the temporal order of events ([Figure 12.2B](#)). This study makes two important points: the first is that even children as young as 3 years old can display episodic-like memory, even if they cannot

always report those memories verbally. Second, the 4-year-olds performed better, indicating that memory for temporal context, at least, continues to grow and mature over this period, along with the ability to express that information verbally.



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FIGURE 12.2 Episodic-like memory in children Just like nonverbal animals, very young children can display memory for what happened where and when, even if they cannot yet verbalize those memories well. (A) Researchers asked 3- and 4-year-old children to hide three stuffed toys around their homes. Later, the 3-year-olds were worse than the 4-year-olds at verbally reporting the what, where, and when of these events. (B) But when allowed to demonstrate their knowledge through behavior (“Can you show me the toy we hid first?”), the 3-year-olds performed as well as the 4-year-olds at remembering what was hidden where, though they were still impaired at remembering the temporal order in which objects were hidden.

Information from [Hayne & Imuta, 2011](#).

Description

Graph A corresponds to verbal recall. The horizontal axis shows What? (Toy) Where? (Room) Where? (Location in room), and When? (Order). The vertical axis shows the number of correct items reported ranging from 0 to 3.5. The data of the graphs are as follows.

What? (Toy). 3-year-olds, 1.3, 4-year-olds, 2.3. Where? (Room). 3-year-olds, 1.9, 4-year-olds, 2.6.

Where? (Location in room). 3-year-olds, 1.5, 4-year-olds, 2.5. When? (Order). 3-year-olds, 1.2, 4-year-olds, 2.3.

Graph B corresponds to behavioral test. The horizontal axis shows Where? (Room) Where? (Location in room), and When? (Order). The vertical axis shows the number of correct seeking behaviors ranging from 0 to 3.5. The data of the graphs are as follows.

Where? 3-year-olds, 2.9, 4-year-olds, 3.1. Where? (Location in room). 3-year-olds, 2.7, 4-year-olds, 3.0. When? (Order). 3-year-olds, 1.0, 4-year-olds, 2.5. All data are approximate.

Sensitive Periods for Early Learning

As you have just read, many types of learning are functional early in life but become more efficient as the organism matures. Sometimes, the opposite is true. In some species, certain kinds of learning are most effective early in life, during a specific time window known as a [sensitive period](#).

sensitive period

A time window, usually early in life, during which a certain kind of learning occurs most easily and is most effective.

Imprinting

One example of a sensitive period is [imprinting](#) (introduced briefly in [Chapter 2](#)). Imprinting refers to the way that animals of many species, including birds, are especially likely to form an attachment to the first individual they see after birth ([Insel & Fernald, 2004](#); [Lorenz, 1935](#)). Normally, the first individual a baby bird sees is its mother or a sibling in the nest, so the chick appropriately imprints on a member of its own species. But, in a classic series of experiments, researcher Konrad Lorenz found that if he removed a newly hatched goose chick from an incubator, the chick would imprint on *him*—and would follow him about as if he were its mother. If the gosling were later placed among a brood of goose chicks following a goose mother, it would ignore the members of its own species in preference for following Lorenz, its human “parent.” Researchers have since shown that chicks can be induced to imprint not only on individuals of the wrong species but even on rotating cylinders and stuffed chickens ([Johnson, 1992](#)). Since Lorenz’s time, imprinting has been demonstrated in a variety of other species, including turkeys, sheep, deer, and buffalo ([Michel & Tyler, 2005](#)).



Thomas D. McAvoy/The LIFE Picture Collection/
Getty Images

Researcher Konrad Lorenz being followed by goslings that imprinted on him because he was the first moving object they saw after hatching. Thereafter, they responded to him as if he were their mother. Why might imprinting normally serve a

useful function for young geese?

imprinting

The tendency of young animals of some species to form an attachment to the first individual they see after birth.

It used to be thought that imprinting was an example of learning that had not merely a sensitive period but a *critical period*—meaning that this type of learning was possible only during a limited time window, and after the window closed, no further such learning could ever occur. Now we know that, at least under some circumstances, the time window for imprinting can be extended, and we also know that imprinting can sometimes even be unlearned ([Thomas & Johnson, 2008](#)). For this reason, most researchers now prefer the term *sensitive period* to refer to time windows when learning is particularly fast or effective, without necessarily claiming that learning at other times will be downright impossible.

Sensitive Periods for Vision and Birdsong

Many types of learning show sensitive periods. [Chapter 3](#) discusses one example: tuning of neurons in the visual cortex. David Hubel and Torsten Wiesel conducted a classic series of studies in which they sewed shut one eye of newborn cats or monkeys and then after several weeks reopened the eye ([Hubel & Wiesel, 1977, 1998](#)). The result? The animals were “blind” in that eye even though they had no physical damage to the visual system. For cats, the period from about 3 weeks to 60 days after birth is a sensitive period for the development of the visual system; for monkeys, the period may last for about the first 6 months of life.

Similar effects are seen in human infants who are born with cataracts that block vision in one eye. If corrective surgery is performed within a few months of birth, vision will develop normally ([Maurer & Lewis, 1999](#)), but if surgery is delayed for a few years, normal vision never develops ([Vaegan, 1979](#)). In one dramatic case, a patient known by the initials M.M. was blinded at age 3½, when a chemical explosion destroyed his left eye and damaged his right eye. For the next 43 years, he had some perception of light but no ability to see form or contrast. At age 46, he underwent a corneal transplant to restore vision in his remaining eye. Ten years later, he showed normal perception of color and motion but continued to have severe deficits in the ability to recognize pictures of objects or identify faces as male or female, happy or sad ([Huber et al., 2015](#)). This is consistent with the idea that high-level visual processing in the brain has a sensitive period early in life, but limited ability to reorganize based on experience during

adulthood.

Song learning in birds provides another example of a sensitive period. [Chapter 11](#) describes how some species of birds learn by listening to and imitating adults of their species. Adult white-crowned sparrows (see the chapter-opening [photo](#)) have a repertoire of about seven different sounds that serve various functions, including territory defense and courtship. Six of these sounds are more or less the same in all white-crowned sparrows, but one—the male song—differs by geographical location. In effect, male sparrows from different regions have different “dialects,” just as people in different regions may speak the same language but have different accents. Normally, a male white-crowned sparrow spends his first 10 days or so in the nest and then moves out to live independently from, but close to, his parents. During the next few months, he will hear his father and other male neighbors sing, and eventually, he himself will begin to sing, using the same “dialect” as they do.

Male sparrows raised in isolation, with no opportunity to hear other adults, begin to sing, too, but their songs are abnormal ([Marler, 1970](#)). Males raised in isolation but allowed to hear tape recordings of male song can learn normally, as long as they hear the song during days 30 through 100 of their life ([Marler, 1970](#)). Earlier or later exposure is of no use: once the sensitive period has passed, the males will not learn normal song.

Language Learning

Some researchers have suggested that human language learning also has a sensitive period, which ends by about age 12 (or earlier). Early evidence for this idea came from the famous and tragic case of “Genie,” a girl whose mentally ill father kept her locked alone in a room, strapped to a potty chair or crib, punishing her if she made any noise ([Curtiss, 1977](#)). The California authorities took Genie away from her parents in 1970, when she was 13 years old. At the time, Genie weighed only 59 pounds and could not straighten her arms or legs nor control her bladder or bowels. She could not speak at all.

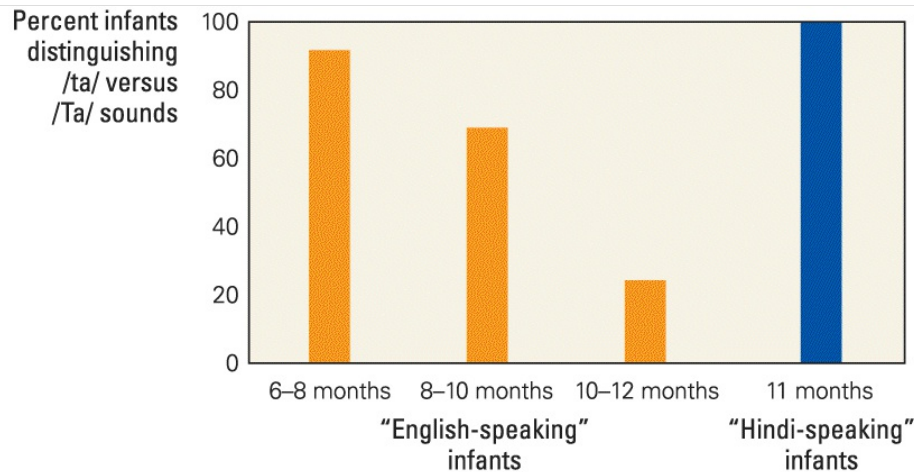
Over the next several years, physicians, psychologists, and social workers worked hard to undo the damage that had been done to Genie. Her health improved greatly, and her IQ appeared to be in the low-normal range. But her language never developed beyond that of a 3- to 4-year-old. She could utter simple sentences (“Want food.”) but not much more. Apparently, Genie’s lack of

exposure to language during the first decade of life left her unable to acquire the complex grammar and vocabulary that characterizes normal adult speech ([Curtiss, 1977](#); [Rymer, 1994](#)). Similarly, her lack of social interactions during development greatly reduced her opportunities to observe and imitate how people use language. An anonymous report from 2008 states that Genie, then age 51 and living in California as a ward of the state, had once again reverted to speechlessness.

We do not have to look at cases as extreme as Genie's to see evidence of a sensitive period in human language learning. In general, children can learn a second language more easily, and approximate a native accent more closely, than adults can ([Newport, 1990](#)). This is partly due to a sensitive period for the perceptual learning that allows phonetic discrimination.

Each human language uses about 25 to 40 speech sounds (called *phonemes*), but different languages may use different sets of sounds. Adults who speak one language can distinguish between phonemes used in their own language but often cannot distinguish between pairs of phonemes from other languages if those sounds are not distinguished in their own language. For example, the phonemes /l/ and /r/ are distinct in English (so that “ray” and “lay” are different words) but not in Japanese; monolingual Japanese adults often cannot distinguish these two phonemes. On the other hand, monolingual English speakers cannot hear the difference between a soft “p” and a sharp “p”—two speech sounds that are meaningfully separate in the Thai language. In effect, the phonemes /r/ and /l/ are part of the same sound category for Japanese speakers, while soft “p” and sharp “p” are part of the same sound category for English speakers.

Infants younger than 6 to 8 months can distinguish all these sounds, even when the distinction does not exist in their native language. This ability to discriminate can be demonstrated by training infants to turn their heads for reinforcement to one sound but not the other. Infants get better at distinguishing speech sounds from their own language as they age but become less able to tell apart sounds that are not distinguished in their own language ([Kuhl, 2000](#)). Thus, a 6-month-old “Japanese-speaking” baby will be able to distinguish “lay” and “ray,” but an 11-month-old one will not. Similarly, 6-month-old “English-speaking” infants can distinguish syllables from Hindi that sound alike to adult English speakers ([Figure 12.3](#)), but this ability is lost by 12 months of age ([Werker & Tees, 1999](#)).



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FIGURE 12.3 Decline in ability to distinguish sounds from other languages At 6 months of age, “English-speaking” infants can easily distinguish the sounds of the Hindi /ta/ versus /Ta/, which are indistinguishable to monolingual English-speaking adults. By about 10 to 12 months of age, this ability is lost in “English-speaking” babies, although 11-month-old “Hindi-speaking” babies can still easily distinguish the two sounds.

Information from [Werker & Tees, 1999](#).

Description

The horizontal axis shows age groups of infants, and the vertical axis shows the percentage of infants distinguishing /ta/ versus /Ta/ sounds. The data of the graph are as follows.

Open quote, English speaking, close quote, infants. 6 to 8 months, 90 percent; 8 to 10 months, 65 percent; 10 to 12 months, 22 percent

Hindi-speaking infants. 11 months, 100 percent. All data are approximate.

These results, together with the tragic case of Genie, suggest that humans acquiring language—like birds acquiring birdsong—have a window of opportunity to learn to distinguish language sounds; beyond that period, learning is possible but much less effective. Children who are fortunate enough to live in a bilingual home and to be exposed to the sounds of multiple languages during this sensitive period are much more likely to grow up to be fluent speakers of those languages than children who must try to learn the sounds later. Early exposure facilitates fluency in gestural languages, such as American Sign Language (ASL), as well as for spoken languages. (For more on the possible relationship between learning spoken and gestural languages, see “[Learning and Memory in Everyday Life](#)”.)

On the other hand, not everyone agrees with the idea of a sensitive period for language learning.

Adults obviously can master second languages, and they often do so more quickly than children, even if their final level of attainment is not as high ([Thomas & Johnson, 2008](#)). Apparently, children learn a new language differently than adults do. The dominant process for children seems to be social imitation; they learn to mimic the speech around them. By contrast, the dominant process for adults involves semantic memory, including explicit strategies such as memorization of new vocabulary ([Hudson Kam & Newport, 2005](#)). As a result, given an equivalent amount of time to learn a second language, adults and older children often make much faster progress than younger children do ([Snow & Hoefnagel-Höhle, 1978](#)).

LEARNING AND MEMORY IN EVERYDAY LIFE

Teaching Babies Signs Before Speech

In the 2004 movie *Meet the Fockers*, the toddler Little Jack uses sign language to inform his grandfather that he'd like a snack and then a nap, after which he'll probably need a diaper change. Just a Hollywood joke about overambitious parenting, right? Well, maybe not. Based on parents' reports, children as young as 6 to 9 months, who have not yet mastered spoken language, can use gestures to communicate a desire for more food or for a bottle of milk ([Acredolo & Goodwyn, 2002](#)). At about 10 to 12 months, children can reach toward an object to indicate that it is wanted or hold an object upward to direct an adult's attention to it ([Goodwyn, Acredolo, & Brown, 2000](#)).

A few months later, children master representational gestures, such as flapping their arms to represent "bird" or "flight" or spreading and unspreading the index and middle fingers to connote "scissors" or "cutting." Many studies show a positive correlation between gesturing and verbal development: the more gestures children learn between 1 and 2 years of age, the larger their verbal vocabularies at age 2 or 3 ([Capone & McGregor, 2004](#); [Goodwyn et al., 2000](#)).

Why might communicating with gestures facilitate the development of verbal language? Susan Goodwyn and colleagues have suggested several possible reasons ([Goodwyn et al., 2000](#)). One is simply that infants learn verbal language more quickly if they are exposed to more vocalizations from their parents. A child who points at a bird in a tree can elicit a parental response ("Yes! That's a birdie!"); a child using 20 such gestures may be able to elicit such responses 10 times more often than a child with only 2 gestures. A second possibility is that babies (like all other creatures) learn best about the things they're interested in. Communicating with gestures may allow the child to show the parent what the *child* is interested in, which then cues the parent to introduce language related to that interest. A third possibility is that communicating with gestures provides a "scaffolding" for the development of verbal language. The child learns a simple gesture that in turn provides insight into how useful communication can be, which motivates the child to explore other ways to communicate, including spoken language.

In the meantime, gestural language is a way for toddlers to communicate their needs when they do not have the ability to express themselves verbally. Goodwyn and her colleagues report an example in which a 14-month-old boy was able to use his "hot" gesture (blowing hard) to let his mother know when his bathwater was too hot, while another toddler signaled "gentle" (petting the back of one hand) to complain that her parent was holding her legs too tightly during a diaper change ([Goodwyn et al., 2000](#)). This isn't quite the sophistication of Hollywood's Little Jack, but it is still impressive for an infant who would otherwise be reduced to frustrated squalling to get her point across.

Adolescence: Crossing from Childhood into Adulthood

Puberty, the process of physical change during which the body transitions to sexual maturity, occurs at different ages in different species. In the United States, girls typically begin the process of puberty around age 10 and have their first menstrual bleeding about 2 years later; first ovulation may occur 1 or 2 years after that, at which point girls are capable of sexual reproduction. Boys typically begin puberty around age 12, and full fertility is gained a few years after that. By contrast, elephants reach sexual maturity at about 9 to 12 years of age, and rats reach sexual maturity by the time they are about 4 to 5 weeks old. In some species, puberty does not occur at a predetermined age. For example, a fully grown female prairie vole remains sexually immature until exposed to scent signals from a male outside her kin group; within 24 hours of this exposure, she becomes sexually receptive, mates, and forms an enduring pair bond with her chosen partner.

puberty

The process of physical change during which the body transitions to sexual maturity, usually beginning around age 10 in human females and age 12 in human males.

Adolescence is defined as the transitional stage between the onset of puberty and entry into adulthood; in humans, this corresponds roughly to the teenage years (ages 13 through 19), although there are wide differences in each individual's physical and social development. Cultural rules also help determine the time frame, and there may be social or religious rites to mark the passage into adulthood. For example, in Western societies, adulthood is associated with the advent of new privileges such as the right to drive, purchase alcohol, and vote; new responsibilities such as leaving home and seeking full-time employment; and new social roles such as marriage and parenthood.

adolescence

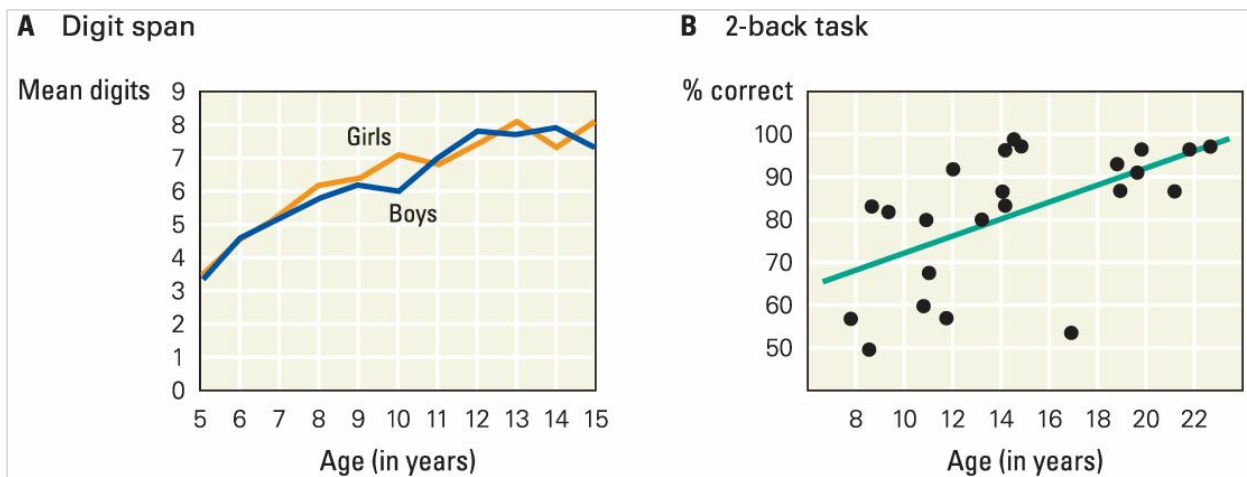
The transitional stage between puberty and adulthood, corresponding roughly to the teenage years in humans (ages 13 to 19).

Thus, puberty is a strictly physical process, while adolescence is a period of psychological and social change. Adolescence and puberty overlap, but whereas puberty has a specific end point (sexual maturity), the boundaries of adolescence are much less precisely defined.

Maturation of Working Memory

Some of the most profound changes during adolescence concern learning and memory abilities. Most prominently, working memory and executive function, which are among the latest learning and memory abilities to mature, continue to develop through adolescence and even into early adulthood.

[Chapter 9](#) defines *working memory* as a short-term storage system that can hold information for active processing. In humans, we often measure working memory capacity in terms of *digit span*, the number of digits that a person can hold in memory at the same time. [Figure 12.4A](#) shows that young children have a relatively short digit span. On average, 5-year-olds can remember and repeat only 3 or 4 digits at a time. But digit span increases through adolescence, so that by age 14 or 15, adolescents can remember approximately 7 digits—the same as adults ([Snow & Hoefnagel-Höhle, 1978](#)). Similar results are obtained if the items to be remembered are visual patterns ([Gathercole, 1998](#)).



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FIGURE 12.4 Working memory in adolescence (A) Digit span, the number of digits a person can hear and then repeat from memory, increases through childhood, reaching normal adult levels (about 7 digits) in late adolescence. (B) Performance on a spatial version of the “2-back” test.

(A) Data from [Gathercole, 1998](#). (B) Information from [Kwon et al., 2002](#).

Description

The graph A depicting digit span shows the age in years from 5 to 15 along the horizontal axis, and the vertical axis has the mean digits from 0 to 9. While girls and boys of age 5 have almost the same digit span of 3.5, the increase is rapid in girls between the ages of 7 and 11. The graph shows that 10-year-old girls have a digit span of 7 while boys of the

same age group show a digit span of 6. Between 11 and 15 years of age, the digit span for boys and girls increases and decreases to reach 7 for boys and 8 for girls.

The scatterplot graph B depicting 2-back task shows the age in years from 8 to 22 along the horizontal axis, and the vertical axis shows the percent correct ranging from 50 to 100. A positive slope starts from (6, 65) and ends at (23, 99). There are many plots plotted above, below, and on the positive slope. Three significant plots are (12, 59), (11, 60), and (17, 55). All data are approximate.

One reason very young children remember fewer numbers and pictures than adults is simply that children have had less exposure to such things. By adolescence, individuals have accumulated much more experience with numbers, words, and patterns, and this knowledge may give them an advantage at encoding those items into working memory. Consistent with this idea is the finding that if we use material the children are highly familiar with, their performance improves. In one clever study, adults and children were asked to remember the locations of chess pieces on a chessboard. Ten-year-old chess experts could remember more pieces than non-chess-playing adults, even though the children's digit spans were lower than the adults' ([Chi, 1978](#)). This suggests that the age-related improvement in working memory capacity at least partially reflects exposure to and familiarity with the material to be remembered.

More complex working-memory abilities mature even later. For instance, [Chapter 9](#) describes the “2-back” task, in which participants are presented with a stream of items; as each new item is presented, participants decide whether it is the same as the one that appeared two items previously. The task can be altered to assess spatial working memory. For example, on each trial, the participant sees an object (such as an “O”) at one of several locations on a computer screen; the participant's job is to press a button whenever the location on the current trial is the same as the location two trials back. The 2-back task requires a great deal of concentration, and even adults don't usually manage to get 100% correct. In one study, the task was administered to participants ranging in age from 7 to 22 years. [Figure 12.4B](#) shows that performance continued to improve through adolescence and into adulthood ([Kwon, Reiss, & Menon, 2002](#)). Other complex working-memory tasks show similar increases throughout adolescence before leveling off at adult levels.

Male–Female Differences in Learning and Memory

Given the dramatic physical changes that occur during adolescence and the fact that most of them are associated with sexual maturation, it is not surprising that many differences between

males and females in learning and memory arise during adolescence. For instance, young adult women often outperform same-age men on tests of language and verbal memory, such as recalling a list or a story, and on some kinds of spatial learning, such as remembering the locations of objects ([Barnfield, 1999](#)). On the other hand, men generally outperform women on other kinds of spatial learning, such as learning the way around a maze ([Astur, Ortiz, & Sutherland, 1998](#)). In one study, men and women were asked to study a map of a fictitious town ([Galea & Kimura, 1993](#)). On average, men were quicker to learn a route within the town, whereas women were better at remembering the positions of landmarks in the town.

In discussing sex differences—or any group differences—it is always important to stress that these are *average* differences. Even though women may outperform men *on average* on a particular task, such as landmark learning, the highest-scoring men will still do better than most of the women (and men). Similarly, even though men may outperform women *on average* on a task of route learning, the highest-scoring women will outscore most of the men (and women). In general, the differences between the highest- and lowest-scoring individuals of either sex are much greater than the average differences between sexes ([Hyde, 2014](#)). It is also important to stress that sex differences in the lab do not necessarily imply differences in the real world. Men may be better—on average—at route learning, whereas women may be better—on average—at navigating based on landmarks, but both methods may be equally effective for finding the way home from the grocery store.

Nevertheless, male–female differences have been shown repeatedly on tasks of spatial learning, language learning, and more ([Cahill, 2005](#); for review, see [Hyde, 2014](#)). One factor contributing to this difference is social influence, including cultural stereotypes in which boys are expected to excel at some kinds of learning while girls are expected to excel at others. But this cannot be the whole story. Differences are also reliably observed between females and males in nonhuman species, and these sex differences cannot easily be attributed to cultural stereotyping. For example, in a radial arm maze, adult male rats are usually better than females at remembering which maze arms never contain any food, and adult female rats are usually better than males at remembering which arms have already been visited on the current day ([Bimonte, Hyde, Hoplight, & Denenberg, 2000](#); [Hyde, Sherman, & Denenberg, 2000](#)).

These differences do not emerge until puberty, however. Thus, male–female differences in

spatial learning appear in sexually mature rats but not in immature ones ([Kanit et al., 2000](#)). Similarly, although adult women outperformed adult men at remembering landmarks in the fictitious town, 8- to 13-year-old girls did no better than same-age boys ([Silverman & Eals, 1992](#)).

Why might these differences be evident only in adult rats and humans? Starting in puberty, there is a dramatic increase in the release of sex hormones, primarily **estrogens** in mature females and **androgens**, particularly **testosterone**, in adult males. The increase in sex hormones occurs at about the same time that many differences appear between males and females in learning and memory, suggesting that the hormones are responsible for many of the behavioral effects. [Section 12.2](#) describes more about the specific ways these sex hormones influence male and female brains.

estrogen

Any member of the principal class of sex hormones present in adult females.

androgen

Any member of the principal class of sex hormones present in adult males.

testosterone

The most important androgen and hence the principal male sex hormone.

The Aging Memory: Adulthood Through Old Age

Most of the learning and memory studies discussed in the other chapters of this book were based on data collected from healthy young adults—whether those adults were humans, rats, chimpanzees, or sea slugs. This is partly because the brain is mature in young adults, so there is less variation among individuals as a result of minor differences in age than is seen earlier in life. (The difference in performance due to age between a 6-month-old and an 18-month-old human is huge, but the difference due to age between a 20-year-old and a 21-year-old is usually minimal.) In fact, most people (barring injury or disease) show relatively stable learning and memory—and other types of cognitive function—throughout adulthood.

For example, the Seattle Longitudinal Study tested more than 6,000 individuals of different ages. Participants were given tests to evaluate several types of cognitive ability and were then re-evaluated at 7-year intervals. As shown in [Figure 12.5](#), the study found relatively little change in

most kinds of cognitive ability—including verbal memory—as participants age from their twenties to their fifties ([Schaie, 2005](#)).

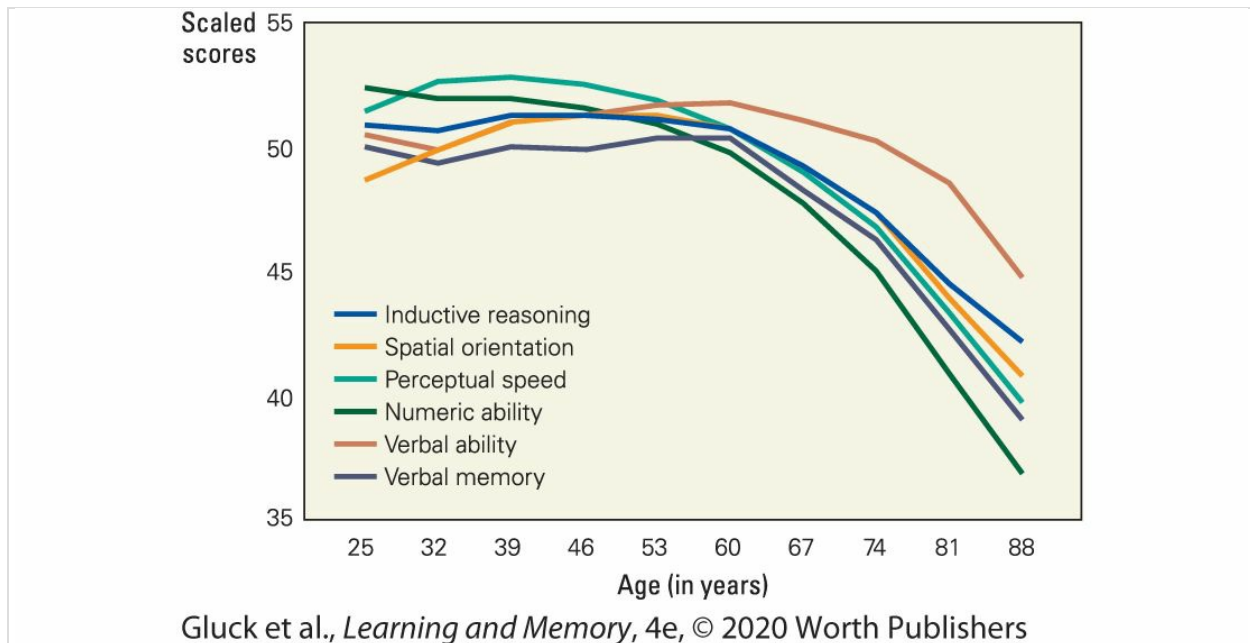


FIGURE 12.5 Human cognition across the lifespan In the Seattle Longitudinal Study, individuals of different ages were tested at 7-year intervals on several types of cognitive ability. Scores have been scaled so that 50 represents the median score observed for the 53-year-old age group in this study; this allows comparisons of data collected from different types of cognitive tests. Most cognitive abilities—including verbal memory—showed little difference in participants from 25 to 60 years of age, but declined in older participants.

Information from [Schaie, 2005](#), Figure 4.

Description

The graph shows age in years from 25 to 88 on the horizontal axis and the scaled scores from 35 to 55 on the vertical axis.

Six lines on the graph represent Inductive reasoning, Spatial orientation, Perceptual speed, Numeric ability, Verbal ability, and Verbal memory. All six lines range between 48 and 53 along the vertical axis, and at 25 years on the horizontal. All six lines remain the same until 61 years, after which the scores decline and range between 47 and 35 years at 88 years. All data are approximate.

Unfortunately, as [Figure 12.5](#) also shows, many cognitive abilities start to decline as humans reach their sixties and beyond. In this and any other discussion of age-related decline, it is important to remember that there are large individual differences in the rates of change, meaning that some people “age well,” while others develop more severe deficits ([Wilson et al., 2002](#)).

However, some general patterns exist. While some forms of memory, such as working memory, start to decline in humans as young as the mid-thirties, others, such as semantic knowledge and verbal ability, tend to remain strong well into old age.

Working Memory in Aging Adults

Working memory, one of the last memory systems to fully mature in humans and other animals, is also one of the first to show deficits in the course of healthy aging. For instance, as discussed previously, healthy young adults have a digit span that averages about 7 digits. However, in elderly adults, the average drops to about 6 to 6.5 digits. This small but significant drop could mean the difference between remembering or forgetting a 7-digit phone number. If the task is made more complicated, such as by presenting different digits simultaneously to each ear, reductions in digit span can be observed as early as age 30 or 40 ([Barr, 1980](#)).

The reason is not yet entirely clear, but one theory suggests that older adults are more susceptible to proactive interference than are younger people. As defined in [Chapter 7](#), *proactive interference* occurs when previously stored information (e.g., a prior address or a computer password) interferes with the ability to remember newer information (see [Figure 7.7](#)). Possibly, older adults are less able to inhibit irrelevant, older information from entering working memory, where it crowds out the information that is relevant now ([Bowles & Salthouse, 2003](#)).

Conditioning and Skill Learning in Aging Adults

In general, learning by conditioning declines with age: older adults are less able than younger adults to adapt their behavior in response to reinforcement. For example, classical conditioning of the eyeblink response starts declining at about age 40 or 50 in humans. Older adults take about twice as long to learn an eyeblink response as younger ones do ([Solomon, Pomerleau, Bennett, James, & Morse, 1989](#); [Woodruff-Pak & Thompson, 1988](#)). There are similar age-related declines in eyeblink conditioning in rabbits ([Solomon & Groccia-Ellison, 1996](#)), rats ([Freeman & Nicholson, 2001](#)), and cats ([Harrison & Buchwald, 1983](#)). Old age does not prevent such learning, but it makes such learning slower.

Skill learning also declines in old age. In [Chapter 8](#), you read about the *rotary pursuit task*, which requires keeping a stylus positioned over a point on a disk that rotates rapidly (see [Figure 8.5](#)). Middle-aged adults can learn this task about as well as young adults, but elderly adults

(over age 60) show a precipitous drop-off in performance ([Ruch, 1934](#)). Real-world skill learning shows the same age-related decline. For instance, one study found that elderly individuals can master the mental and physical skills needed to use computers, but their rate of acquisition is slower than that of younger learners, and they make more errors during learning than younger learners do ([Czaja, Hammond, Blascovich, & Swede, 1993](#)). Similarly, aged rats, monkeys, and other animals tend to learn new motor skills more slowly than younger animals do ([Bachevalier et al., 1991](#); [Fahlström, Yu, & Ulfhake, 2011](#)). In short, you *can* teach an old dog new tricks; it just takes a while longer.

On the other hand, even though the learning of new associations and skills is slowed in healthy aging, highly practiced skills tend to be maintained well. Famous examples are Spanish cellist Pablo Casals, who continued to compose music and conduct orchestras into his nineties, and Jack Nicklaus, who continued to win major golf tournaments in his forties and fifties—besting many younger players. Expert typists in their sixties and seventies can often execute their skill as quickly and accurately as typists in their twenties and thirties ([Salthouse, 1984](#)). Many expert chess and bridge players improve, rather than decline, with age, and airline pilots ages 40 to 60 actually have fewer accidents than younger pilots ([Birren, 1964](#)), all in support of the axiom that age and experience can often defeat youth and speed.

Episodic Memory and Semantic Memory: Old Memories Fare Better than New Learning

Existing episodic and semantic memories tend to survive very well even into extreme old age. [Chapter 7](#) notes that semantic memory is normally strengthened by repeated exposure. By the time a person reaches old age, semantic information such as vocabulary and general knowledge about the world has been encoded, retrieved, and re-encoded many times. Healthy elderly adults generally experience little or no drop-off in the ability to retain and retrieve such semantic memories ([Light, 1991](#)). Older individuals also tend to show only a modest drop-off in their ability to recall episodic memories from their distant past ([Piolino, Desgranges, Benali, & Eustache, 2002](#)).

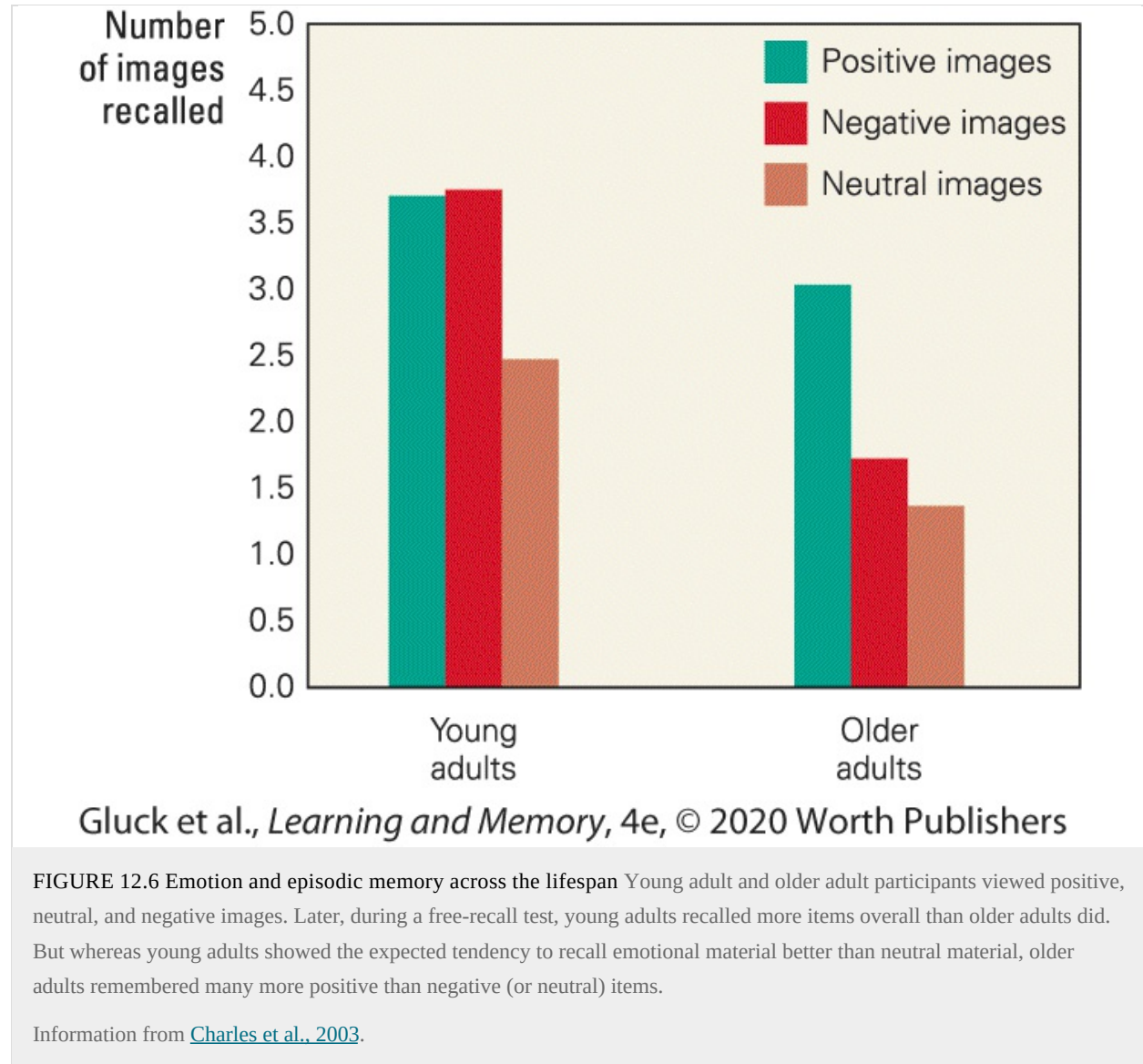
But although well-formed memories may survive, elderly adults are generally less effective at forming new episodic and semantic memories. This is reflected in [Figure 12.5](#) by participants' decline in "verbal memory," measured via free-recall tests for a short story and for a list of

words. Elderly adults also fare much worse than young adults on tests of *paired-associate learning*, in which participants first study a list of word pairs and are then given the first word of each pair and asked to recall the second member of the pair ([Canestrari, 1963](#)). The deficit in elderly adults appears to be due to encoding difficulties rather than retrieval difficulties. Thus, for example, elderly adults' performance at paired-associate learning is much improved if items are presented at a slower pace during study, presumably because the elderly adults now have more time to encode the information ([Canestrari, 1963](#)). Elderly adults' performance also improves if the studied items have some meaning to them ([Graf, 1990](#)). For instance, in one study, college-age adults outperformed elderly adults on memorizing a list of names of current rock stars, but the elderly adults outperformed the students on memorizing a list of musicians who had been popular with the earlier generation ([Hanley-Dunn & McIntosh, 1984](#)).

Aging also affects directed forgetting. As discussed in [Chapter 7](#), in *directed-forgetting* tasks, participants are asked to study information and then to intentionally forget some of the items (see [Figure 7.6B](#)). When participants are given a surprise memory test, they tend to recall fewer of the items they were specifically instructed to forget than items they were supposed to remember (or items for which they received no forgetting instructions). Under these conditions, younger adults can intentionally forget *more* items than elderly adults ([Titz & Verhaeghen, 2010](#)). In short, as we age, we seem to have less control over what gets encoded; we become less able to store the items we do want to keep and simultaneously less able to discard those items we wish to forget. In support of the idea that aging compromises our control over memory retrieval, a recent study found that older adults could greatly improve their success on a directed-forgetting task if they received focused instructions about how to inhibit unwanted retrieval ([Murray, Anderson, & Kensinger, 2015](#)).

Emotion may also affect memory differently as we age. [Chapter 10](#) discusses how the emotional content of information can affect how strongly that information gets encoded in memory. Some studies suggest that older adults are more likely than young adults to remember positive information. In one such study, participants viewed a series of positive images, such as smiling children and flowers; negative images, such as snakes and injured bodies; and neutral images, such as inanimate objects ([Charles, Mather, & Carstensen, 2003](#)). On a subsequent free-recall test, young adults recalled significantly more images overall than elderly adults did ([Figure 12.6](#)). In addition, the nature of the recalled images differed. The young adults recalled about

equal numbers of positive and negative images but fewer emotionally neutral images. This is consistent with the finding that emotion tends to enhance memory encoding (as discussed in [Chapter 10](#) and shown in [Figure 10.6](#)).



Description

The graph shows two age groups on the horizontal axis and the number of images recalled ranging from 0 to 5 on the vertical axis.

The data of the graph are as follows. Young adults: positive images, 3.7; negative images, 3.71; neutral images, 2.45. Older adults: positive images, 3.0; negative images, 1.7; neutral images, 1.4. All data are approximate.

In contrast, the older adults remembered many more positive than negative (or neutral) images. One possible explanation is that younger participants process the negative images more deeply than older participants do—either by conscious choice, or due to age-related changes in the brain structures (e.g., the amygdala) that mediate emotional processing, or both. And as described in [Chapter 7](#), according to the *levels-of-processing* principle, deeper encoding during learning tends to improve memory at recall.

Metamemory and Aging

Given the findings described above, you may be surprised to learn that the most common memory-related complaints among healthy elderly individuals are not usually failures to learn new information but failures to retrieve old information on demand. [Chapter 7](#) discusses “tip-of-the-tongue” (TOT) experiences, which occur when people feel sure they know information but cannot retrieve it at the present time. TOT events are among the most frequently reported and frustrating memory problems of healthy adults aged 64 to 75 years ([Sunderland, Watts, Baddeley, & Harris, 1986](#)).

The defining feature of tip-of-the-tongue events is the strong *feeling of knowing* (FOK), which in turn is a feature of *metamemory*, our knowledge and beliefs about our own memory (also discussed in [Chapter 7](#)). Some studies suggest that metamemory becomes less accurate with age, and the age-associated increase in TOT events may not represent failures of memory retrieval so much as failures to correctly assess what information is available ([Salthouse & Mandell, 2013](#)).

Consistent with this idea, one study asked older and younger adults to predict how well they would remember information on a later test. Whereas the younger adults were fairly accurate in predicting how well they would perform, the older adults were highly overconfident, predicting that they would remember much more than they actually did ([Soderstrom, McCabe, & Rhodes, 2012](#)). In summary, then, the age-related decline in acquiring new memory appears to be accompanied by an age-related decline in the ability to accurately assess what we already know. On the other hand, at least one study suggests that older adults are better able to correct their own factual mistakes, perhaps because the older adults are better able to rally their attention after an error ([Metcalf, Casal-Roscum, Radin, & Friedman, 2015](#)).

Learning and Memory in Old Age

Learning and memory abilities generally peak in young adulthood and may decline thereafter. Some types of learning and memory may begin to decline in middle age, while others may remain relatively robust through healthy old age. See if you can identify the learning and memory processes involved in each of the tasks described below and predict whether or not healthy older people should be able to perform them as well (or nearly as well) as when they were young adults. ([Answers appear at the end of the chapter.](#))

1. Recalling one's wedding day
2. Remembering the items on this week's shopping list (without writing them down)
3. Remembering how to make coffee
4. Learning the name of a new friend
5. Learning how to take photos with a new phone

Interim Summary

- An individual's learning and memory abilities vary across the lifespan.
- Some simple learning, such as habituation and recognition, can occur even before birth.
- Most types of learning and memory ability are present in at least a rudimentary form from a very early age, although they may become more efficient as the individual grows toward adulthood.
- Even before young children are able to verbalize their episodic memories, they show behavioral evidence of episodic-like memory, similar to the what-where-when memories that can be demonstrated in nonhuman animals.
- Sensitive periods are windows of time, usually early in life, when particular kinds of learning are easiest or most effective. In humans, there are sensitive periods for language learning as well as for sensory systems, such as vision.
- Puberty is the process of physical change during which the body transitions to sexual maturity. Adolescence is the transitional stage between puberty and adulthood.
- During puberty, there is a dramatic increase in release of sex hormones (primarily estrogens in females and androgens, particularly testosterone, in males), which may contribute to differences in average learning and memory abilities between males and females.
- Working memory is one of the last systems to fully mature in humans, and it might not be fully developed until early adulthood.
- Learning and memory abilities peak in young adulthood but generally decline in healthy aging. A rule of thumb is that old memories may survive well, but new learning may be slowed in aging individuals.
- Age-related declines in metamemory may contribute to frequent and frustrating tip-of-the-tongue (TOT) events.

12.2 Brain Substrates

Unsurprisingly, the age-related waxing and waning of learning and memory abilities reflect underlying changes in the brain across the lifespan. This section describes those changes, starting with the genetic biases laid down before birth, continuing with development in infancy and childhood, looking at the profound changes associated with adolescence and puberty, and finally exploring the changes that occur through adulthood and into old age.

The Genetic Blueprint Is Modified by Experience

Starting at the moment of conception, aspects of an organism's learning and memory abilities are encoded in its genetic structure and begin to guide its future development. The influence of genes on brain function is still a relatively new area of study, but researchers are beginning to understand some of the ways in which genes affect learning and memory.

Genetic Variation and Individual Differences in Learning Abilities

DNA (or, more formally, *deoxyribonucleic acid*) is the material found within the nucleus of all cells that carries the instructions for making a living organism. In plants and animals, DNA is organized into packets called **chromosomes**. Humans have 23 pairs of chromosomes. Chromosomes can be further subdivided into genes. Each **gene** is simply a segment of DNA that contains the molecular instructions, or “recipe,” for building a protein from amino acids. In total, humans probably have about 20,000 to 25,000 genes ([International Human Genome Sequencing Consortium, 2004](#)). In animals, most genes come in pairs (one on each chromosome), one inherited from the mother and one from the father. (Exceptions include egg and sperm cells, each of which contains 23 unpaired chromosomes.)

DNA

(formally, *deoxyribonucleic acid*) The genetic material found within all living cells that contains the instructions for making an organism.

chromosome

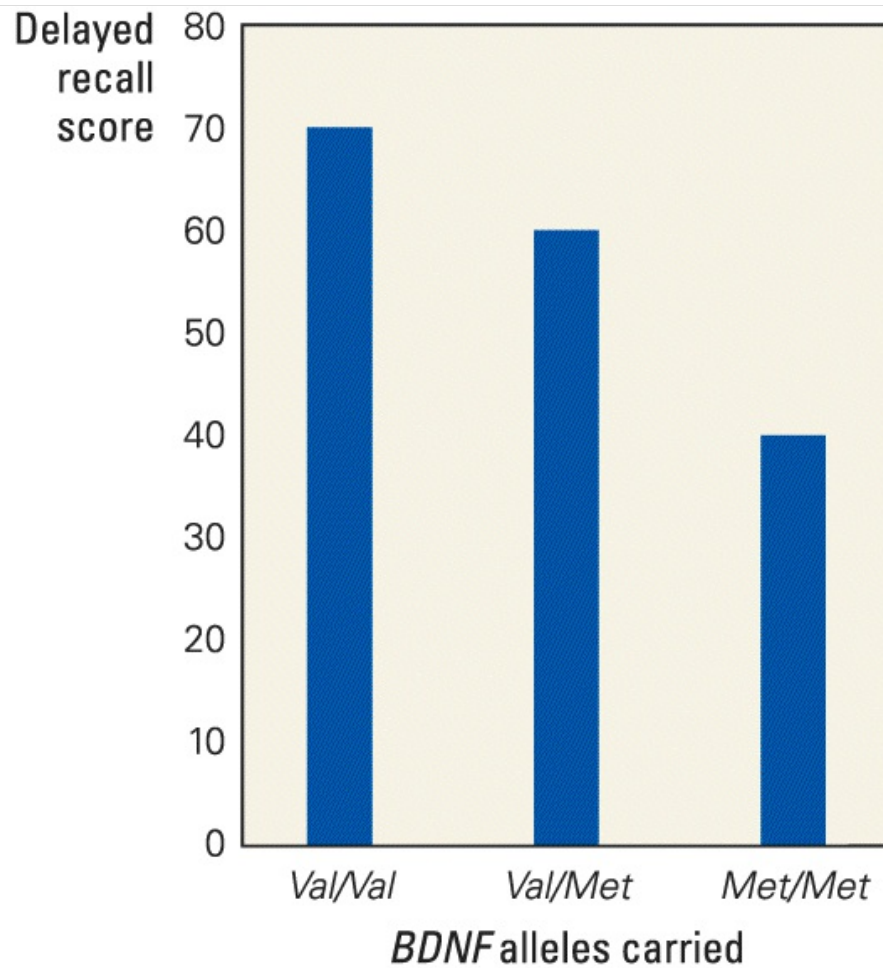
A packet of DNA; most human cells contain 23 pairs of chromosomes, with one member of each pair inherited from the mother and one from the father.

gene

A segment of a DNA strand that contains information for building a protein from amino acids; in essence, genes are the recipes

for constructing and maintaining an organism.

Different genes serve different functions. For example, the *BDNF* gene on human chromosome 11 helps regulate production of brain-derived neurotrophic factor (BDNF), a protein that is vital for the health of neurons. Among other functions, BDNF appears to affect learning and memory by enhancing *long-term potentiation*, or LTP ([Lu & Gottschalk, 2000](#); [Poo, 2001](#)). The *BDNF* gene comes in several naturally occurring variants, or [alleles](#). The most common version of the *BDNF* gene is called the *Val allele*. However, about one-third of people inherit at least one copy of a different version, the *Met allele*, which produces a less effective form of BDNF. People carrying one or two copies of the *Met* allele are slightly worse at learning and memory tasks than people carrying two copies of the *Val* allele. [Figure 12.7](#) shows performance on a test that requires participants to listen to short paragraphs and then repeat them from memory 20 minutes later. People with two copies of the *Val* allele (*Val/Val*) perform better than those with two copies of the *Met* allele (*Met/Met*); people with one copy of each allele (*Val/Met*) are intermediate in performance ([Egan et al., 2003](#)).



Gluck et al., *Learning and Memory*, 4e,
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FIGURE 12.7 Genetic influences on learning and memory in humans BDNF (brain-derived neurotrophic factor) is a protein needed for neuron health and function. People carrying two copies of the *Met* allele of the *BDNF* gene (*Met/Met*) show worse performance on a test of memory recall than people carrying two copies of the *Val* allele (*Val/Val*). People carrying one copy of each allele (*Val/Met*) perform at intermediate levels.

Information from [Egan et al., 2003](#).

Description

The horizontal axis shows the type of B D N F variants in a select group of people and the vertical axis shows their delayed recall score (ranging from 0 to 80) in a test performance.

The data of the graph are as follows. Val-Val, 70. Val-Met, 60. Met-Met, 40.

allele

A naturally occurring variant of a gene.

The *BDNF* gene is only one of many genes that appear to influence learning and memory behavior. Other examples include the *5-HT2AR* gene, which encodes the instructions for building a particular kind of receptor for the neurotransmitter serotonin and therefore may help determine the effectiveness of transmission at certain synapses ([de Quervain et al., 2003](#)); the *WWC1* (or “*KIBRA*”) gene, which appears to help modify synaptic plasticity and may be associated with the rate at which we forget new information ([Schneider et al., 2010](#)); and the *SNC1A* gene, which appears to govern how action potentials propagate down the axon, determining whether the message gets passed on to the next neuron ([Papassotiropoulos et al., 2009](#)). Like the *BDNF* gene, each of these has several naturally occurring variants, and humans who carry different alleles of these genes have slight but significant differences in their ability to learn and recall new information. There are doubtless many more genes that influence memory, and they probably interact with each other and with the environment in complex ways that we are only beginning to understand.

Selective Breeding and Twin Studies

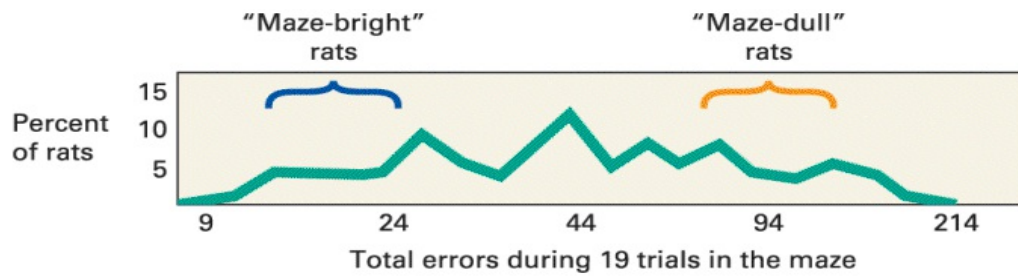
Even without understanding the function of every gene, humans manipulate genes through selective breeding for agricultural and other purposes. Since ancient times, humans have bred animals to obtain particular characteristics: winning racehorses mated together to produce ever-faster offspring, purebred dogs mated together to produce ever-better examples of the breed, and sheep mated together to produce thicker or finer wool. In each case, the goal is for each parent’s “desirable” genes to be passed on to the offspring; by mating two animals with desirable characteristics, the breeder maximizes the likelihood that offspring will inherit those characteristics.

An early experiment in the genetics of learning and memory considered whether animals could be bred for learning ability ([Tryon, 1940](#)). Psychologist Robert Tryon trained a large group of rats in a complex maze; due to individual differences, some rats (which he called “maze-bright”) learned quickly, whereas others (“maze-dull”) learned more slowly ([Figure 12.8A](#)), making many errors (entries into blind alleys) before reaching the goal. Tryon then paired off the maze-bright rats and allowed them to breed; he also bred together the maze-dull rats. When he trained the two groups of resulting offspring in the maze, some offspring from each group again learned better than their peers and some learned worse than average. He repeated the process of mating maze-bright rats together and mating maze-dull rats together, and in this second generation of

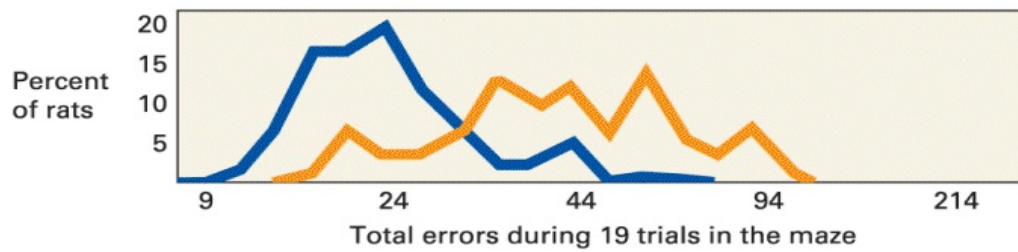
selective breeding, rats descended from maze-bright parents and grandparents tended to outperform the descendants of maze-dull rats ([Figure 12.8B](#)).



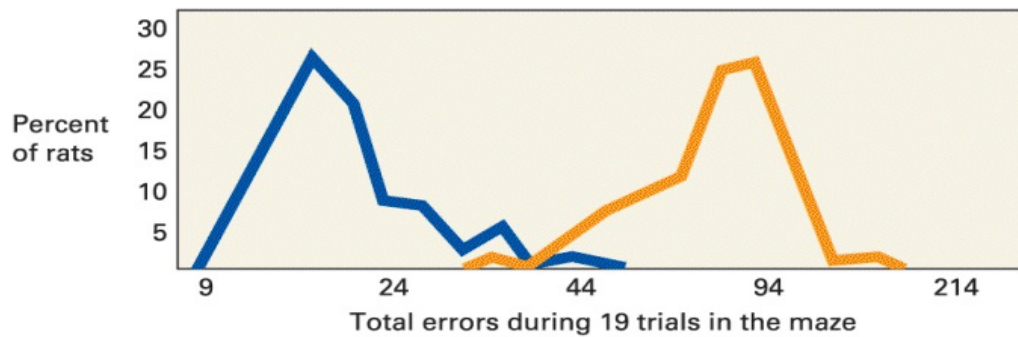
A Original rats



B Second selectively bred generation



C Seventh selectively bred generation



D Effects of environment

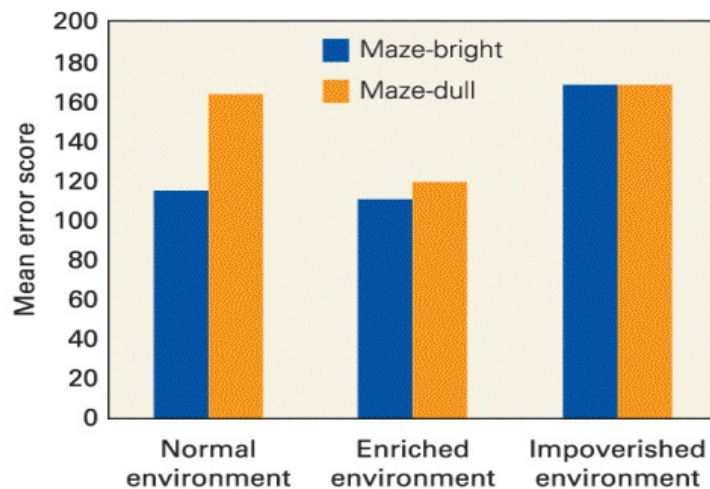


FIGURE 12.8 Breeding smart rats (A) Psychologist Robert Tryon trained rats in a maze ([Tryon, 1940](#)): some (the “maze-bright” rats) made few errors before mastering the task, while others (the “maze-dull” rats) made many errors. (B) Tryon bred the maze-bright rats together and trained their offspring in the maze; he did the same with the offspring of maze-dull rats. By the second selectively bred generation, the descendants of maze-bright rats (blue line) tended to make fewer errors than the descendants of maze-dull rats (orange line). (C) By the seventh generation, rats born into the maze-bright strain routinely outperformed rats from the maze-dull strain. (D) When rats from Tryon’s breeding program were housed under normal laboratory conditions, those from the maze-bright strain made fewer errors in the maze than rats from the maze-dull strain. But rats from the maze-dull strain that were housed in an enriched environment performed just as well as rats from the maze-bright strain; conversely, rats from the maze-bright strain that were housed in an impoverished environment performed as poorly as rats from the maze-dull strain, showing the strong effects of environment on learning and behavior.

(A–C) based on data presented in [Tryon, 1940](#), Figure 4. (D) “Normal environment” data from [Rabinovitch & Rosvold, 1951](#); “enriched environment” and “impoverished environment” data from [Cooper & Zubek, 1958](#).

Description

Graph A corresponds to data on original rats. It has the total errors during 19 trials in a maze along the horizontal axis and the percentage of rats on the vertical axis. Open quotes Maze-bright close quotes rats made about 25 errors during the earlier stages while increased numbers such as 94 and 214 were made by more than 5 percent of open quotes maze-dull close quotes rats.

Graph B corresponds to data on the second selectively bred generation. It shows total errors during 19 trials in the maze on the horizontal axis and the percent of rats on the vertical axis. In the beginning stages, 10 percent of the second generation of the maze-bright rats made about 19 errors and 20 percent of the rats made over 24 errors. As the trials increased the percent of rats making errors declined significantly. On the other hand, a comparatively larger percentage of the second generation of open quotes maze-dull close quotes rats made over 30 errors in the beginning stages and as it progressed, 15 percent of these rats made over 60 errors indicating that the second-generation of maze-bright rats performed better.

Graph C corresponds to data on seventh selectively bred generation, it shows total errors during 19 trials in the maze on the horizontal axis and the percentage of rats on the vertical axis. As much as 25 percent of the seventh generation of open quotes maze-bright close quotes rats made about 20 errors during the early stages of the trials, and with time the percent of rats that made up to 44 errors were less than 5 percent. The maze-dull rats that made over 90 errors during the initial trials accounted for about 25 percent, and the number of errors increased to 118 with time again indicating that open quotes maze-bright close quotes rats outperformed open quotes maze-dull close quotes rats in the younger generations.

Graph D shows the effects of environment on the performance of two categories of rats. The horizontal axis shows the types of environment and the vertical axis shows the mean error scores. The data of the graph are as follows.

Normal environment, maze-bright, 119. maze-dull, 161. Enriched environment, maze-bright, 110. maze-dull, 120. Impoverished environment, maze-bright, 166. maze-dull, 166. All data are approximate.

Tryon repeated the process over and over, breeding together the best of the maze-bright offspring and also breeding together the worst of the maze-dull offspring. By the seventh generation of

offspring, there was almost no overlap: rats from the maze-bright line routinely outperformed rats bred from the maze-dull line ([Figure 12.8C](#)). Later experiments have shown that many different species, including rats, mice, and fruit flies, can similarly be bred for ability on a variety of learning tasks ([Tully, 1996](#)).

Presumably, “maze-bright” and “maze-dull” rats inherit a package of genes from their parents that contribute multiple characteristics resulting collectively in good or poor performance in a maze. These genes could be modifying learning indirectly rather than directly. For example, maze-bright rats may be less anxious, or more prone to explore, or more active overall—and any of these characteristics could translate into better performance in a maze. In fact, learning ability, like all facets of intelligence, appears to be determined by multiple genes interacting rather than by any single gene.

And what about inherited learning abilities in humans? Ethical concerns obviously preclude experiments in which pairs of “smart” or “dumb” people are made to breed with each other to see what kinds of offspring result. However, we can get some clues from twin studies. In these studies, researchers compare the mental abilities of identical twins (who have identical genetic makeup) and fraternal twins (who share 50% of their genes, on average, just like any other siblings). Identical twins show more similarity than fraternal twins on several kinds of learning and memory measures, including tests of working memory (e.g., digit span) and memory for verbal and picture information ([Finkel, Pedersen, & McGue, 1995](#); [Swan et al., 1999](#)). This suggests that genes play a strong role in determining our learning and memory abilities. Some studies of human twins suggest that over half of the variation in individuals’ memory scores may be accounted for by differences in those individuals’ genetic makeup ([McClearn et al., 1997](#); [Swan et al., 1999](#)).

Environmental Interactions

If genes account for about half of the individual variability in learning and memory abilities, then the rest of the variation must be accounted for by nongenetic factors, such as health, stress, living conditions, and social interactions.

In a vivid illustration of this principle, researchers took rats from Tryon’s maze-bright and maze-dull strains and tested them in the maze. [Figure 12.8D](#) shows that, under normal conditions, rats

from the maze-dull strain made more errors than rats from the maze-bright strain, just as you would expect ([Rabinovitch & Rosvold, 1951](#)). But some rats from each strain were housed in an enriched environment, with decorations on the walls, mirrors and balls to play with, and ramps and tunnels to explore. As [Chapter 2](#) discusses, housing in such an enriched environment can affect both brain structure and learning abilities. Sure enough, maze-dull rats that had been housed in an enriched environment performed just as well as maze-bright rats ([Cooper & Zubek, 1958](#)). Other rats from each strain were housed in an impoverished environment, in bare cages containing nothing except the food tray and water cup. Now, the maze-bright rats performed as poorly as the maze-dull rats ([Cooper & Zubek, 1958](#)). Thus, the animals' genetic predispositions could be modified by environmental experiences.

The effects of early exposure to an enriched environment have since been demonstrated across a broad spectrum of species, from rodents to primates, and on a range of mental domains, from maze learning to fear conditioning to object recognition (for review, see [Clemenson, Deng, & Gage, 2015](#)). Presumably, the early experiences help guide brain development, producing more numerous and more densely connected neurons (as shown in [Figure 2.6](#)) that form a rich scaffold to support learning throughout life.

Conversely, severe environmental deprivation during rearing can have the opposite effects, as seen in the rats raised in the restricted environment of [Figure 12.8D](#). Humans show a similar pattern. [Section 12.1](#) describes Genie, raised in a tragically restricted and stressful environment, who never recovered sufficiently to lead a normal adult life. Infants subjected to severe environmental deprivation, such as rearing in low-quality orphanages with insufficient social interaction, show severe delays in cognitive and social development ([Sonuga-Barke et al., 2017](#)) as well as brain changes that may include both reduced numbers and reduced connectivity of neurons in the brain ([McLaughlin, Sheridan et al., 2014](#)). The effects of severe early-life deprivation can persist into adulthood, even if the children are later adopted into supportive, caring families ([Sonuga-Barke et al., 2017](#)).

Such examples of early-life enrichment and deprivation in rats and humans represent extreme cases, but they illustrate how the environment—early in life and at every point thereafter—affects the brain and its function. Genes are the basic blueprint for an individual's learning and memory abilities, but the final outcome reflects an interaction of heredity and environment

([Manuck & McCaffery, 2015](#)). For more on one way environmental conditions may—or may not—influence cognitive abilities, see “[Learning and Memory in Everyday Life](#)”.

LEARNING AND MEMORY IN EVERYDAY LIFE

Can Exposure to Classical Music Make Babies Smarter?

Walk through any children’s store, and you are likely to find shelves of music, books, and videos dedicated to the premise that children who are exposed to classical music will reap intellectual benefits. It all started back in 1993, when physicist Gordon Shaw and developmental psychologist (and concert cellist) Francis Rauscher reported that college students who had listened to 10 minutes of a Mozart sonata before taking an intelligence test scored about 8 or 9 points better than students who sat quietly during the same interval ([Rauscher, Shaw, & Ky, 1993](#)).

The media quickly picked up the story of the “Mozart effect.” Entrepreneurs rushed to produce intelligence-enhancing music products for babies, and well-intentioned parents rushed to buy them. Pregnant mothers spent afternoons playing sonatas to their unborn children, the governor of Georgia recommended that the state provide recordings of classical music to the parents of every newborn citizen, and Florida passed a law requiring all state-funded educational programs to play classical music every day for children under 6.

But, in fact, scientific evidence for the Mozart effect is mixed at best. Although some studies replicated the original findings ([Rideout, Dougherty, & Wernert, 1998](#); [Wilson & Brown, 1997](#)), others did not ([Bridgett & Cuevas, 2000](#); [McCutcheon, 2000](#); [McKelvie & Low, 2002](#); [Steele, Bass, & Crook, 1999](#)). Most researchers now conclude that any general claim of intellectual improvement following mere exposure to classical music is unwarranted ([Chabris, 1999](#); [Fudin & Lembeissis, 2004](#); [Steele et al., 1999](#)). Rauscher and Shaw themselves stressed that their original paper only found an effect of classical music on very specific tasks involving abstract reasoning and mental imagery, such as imagining how a piece of paper will look when unfolded after several steps of folding and cutting ([Rauscher & Shaw, 1998](#)). And there is no evidence that this effect lasts more than 10 to 15 minutes.

It is true that listening to complex music activates the same brain regions that are used in abstract spatial reasoning. Music may “prime,” or prepare, those brain regions so that they are more efficient on subsequent spatial-reasoning tasks ([Rauscher & Shaw, 1998](#); [Rauscher et al., 1993](#)). Listening to music can also produce changes in mood and arousal that might affect performance ([Chabris, 1999](#); [Steele, 2003](#); [Thompson, Schellenberg, & Husain, 2001](#)). However, such short-term effects would not cause long-term improvements in intelligence.

And what about those parents who want a quick way to make their children smarter? Spending large amounts of money on Mozart-for-babies products may not be the answer. At best, such exposure results in a small, temporary increase in a specific kind of spatial ability. More broadly, one review noted that there are more than 80,000 “educational apps” available in Apple’s App Store alone, mostly unregulated and untested, while numerous scientific studies have failed to show that babies are learning effectively from these products ([Hirsh-Pasek et al., 2015](#)). For now, at least, there is no quick trick for turning Baby into Einstein.

Epigenetics

Genes contain the “recipes” for building and maintaining organisms, but they must be activated,

or “expressed,” in order to take effect. You may have a great recipe for chocolate cake, but it is only when you buy the ingredients, mix the batter, and bake the cake that you actually get to taste the results.

Taking the analogy further, an experienced cook is likely to have a large recipe file from which to select appropriate items for tonight’s menu. If she is planning a Cinco de Mayo celebration, she may use the recipes for tacos and enchiladas rather than the Thanksgiving-themed recipes for turkey and mashed potatoes. Similarly, our full complement of genes represents the recipe file for our bodies, and (almost) every cell in our bodies contains this file. So there must be mechanisms (“chefs”) that determine which specific genes (“recipes”) are activated in which cells and at which times, so that cells in the liver, heart, skin, brain, and elsewhere can each develop as needed for that particular organ to perform its special function.

The emerging field of [epigenetics](#) is the study of mechanisms by which gene activation can be modified, without modifying the genes themselves. One such mechanism is *methylation*, the attachment of specific groups of atoms (called methyl groups) to a gene to “tag” that gene and alter (usually inhibit) its activity.

epigenetics

The study of mechanisms by which gene expression can be modified (without modifying the genes themselves).

Whereas our genes are inherited at the moment of conception, epigenetic mechanisms are more flexible. They react to outside factors such as diet, environmental chemicals, and stress. For example, many forms of cancer are caused by genes that are normally suppressed by methylation; if methylation is decreased, these genes may be activated, triggering cancer. An important area of modern cancer research involves drugs that target epigenetic processes to restore a proper balance of inhibited and activated genes.

Epigenetic mechanisms also play a key role in learning and memory ([Day & Sweatt, 2010](#); [Lockett, Wilkes, & Maleszka, 2010](#)). For instance, [Chapter 4](#) describes how memories can be encoded by long-term structural changes in neurons, as when the creation of new synapses is triggered by the activation and inactivation of genes such as *CREB-1* and *CREB-2*. This is an example of epigenetic modulation. The *BDNF* gene, which we discussed earlier in this section and which appears to promote LTP, is also subject to epigenetic influence; specifically, both

acute and chronic stress can lead to reduced *BDNF* activation, which may be one way that stress can impair memory ([Mitchellmore & Gede, 2014](#)). In all, thousands of genes are subject to epigenetic influence at specific times and in specific neurons, triggering processes such as the synaptic changes that affect memory ([Lockett et al., 2010](#)).

One of the most striking features about epigenetic changes is that in some cases, they, like genes, can be passed from parent to offspring. Thus, a mouse that experiences early-life stress will show epigenetic changes—and its own offspring may inherit the same epigenetic pattern, even if the offspring never experience the stressor and never meet their biological parents (for review, see [Dias, Maddox, Klengel, & Ressler, 2015](#)). In humans also, epigenetic changes can sometimes be inherited, such that individuals subjected to extreme stress (e.g., survivors of the Holocaust or the Rwandan genocide) may experience epigenetic changes and may pass along those changes to their children (for review, see [Dias et al., 2015](#)).

Some provocative studies suggest that epigenetics may even provide a mechanism for passing specific learning skills across generations. For example, in one study, male mice were classically conditioned to associate a particular odor with foot shock; these mice developed a fear response to that odor and also showed cortical remapping that resulted in an increased number of olfactory neurons responding to that odor. The offspring of these mice also showed enlargement of those cortical regions, along with enhanced ability to learn about that specific odor ([Dias & Ressler, 2010](#)). Studies like this raise the intriguing possibility that the “smart offspring” of “smart rats,” like those in [Figure 12.8](#), might have inherited not only genetics but also epigenetics that contribute to the descendants’ success in the maze their ancestors learned to run.

Neurons and Synapses in the Developing Brain

As you just read, our genetic inheritance lays down the blueprint for brain function. From this foundation, the developing brain, in interacting with our surroundings and responding to our experiences, produces the neurons and synapses that shape our abilities. This process begins before birth and continues throughout childhood and into adolescence.

Early Overproduction of Neurons

Before birth, the brain develops with amazing speed; at certain points in human gestation, up to 250,000 new neurons are added *each minute* ([McClearn et al., 1997](#); [Swan et al., 1999](#)). The production of new neurons is called **neurogenesis**. By about 25 weeks after conception, the majority of the human fetus's neurons are in place. At this point, the lower-brain centers responsible for such functions as breathing, digestion, and reflexes are almost fully developed.

neurogenesis

Creation of new neurons in the brain.

The process of neurogenesis is not uniform throughout the brain, however. For example, Purkinje cells in the cerebellum are one class of neuron that form relatively early during gestation ([Sidman & Rakic, 1973](#)). This helps explain why classical eyeblink conditioning, which depends on the cerebellum, is already possible in very young infants. But the cerebellum continues to develop after birth, too, which may be why conditioning can occur faster, and under more complicated circumstances, in older children and adults than in infants ([Herbert, Eckerman, & Stanton, 2003](#)).

Surprisingly, after the prenatal flurry of neurogenesis, the infant brain undergoes a period of reduction in the number of neurons. Normally, neurons require compounds called *neurotrophic factors* that help them grow and thrive. BDNF, described earlier, is one example of a neurotrophic factor. Neurons obtain neurotrophic factors from their neighbors. When a neuron is deprived of neurotrophic factors, genes become active that cause the neuron to die. Such natural cell death is called **apoptosis**, to distinguish it from cell death caused by accident or disease. In a sense, apoptosis implements the brain's version of Darwinian natural selection: if many neurons are competing for a limited amount of neurotrophic factor, only some can survive. Neurons that are densely connected to their neighbors, and thus probably play vital roles in brain function, may be more likely to obtain neurotrophic factors and win the competition. Neurons that have less contact with their neighbors—and thus probably contribute less to overall brain function—may be more likely to die through apoptosis.

apoptosis

Natural cell death, as opposed to cell death caused by accident or disease.

During childhood, apoptosis may cull as many as one-third of all the neurons produced prenatally. This may seem like a roundabout way to build a brain: creating billions of neurons

and then destroying a large number of them soon after. But the process allows for a great deal of fine-tuning after birth. The brain starts off with plenty of resources, in the form of neurons, and experience determines which of those resources are critical and which are not so necessary.

Pruning of Synapses

In the same way that the brain starts life with an oversupply of neurons, it also begins with a surplus of synapses. The creation of new synapses, called [synaptogenesis](#), begins in the human brain as early as 5 months after conception. But after birth, synaptogenesis really gets going, with as many as 40,000 synapses being created *per second* in the infant macaque monkey! Like neurogenesis, synaptogenesis occurs at different rates in different brain areas. For instance, in humans, the bulk of synaptogenesis in the visual cortex is completed by about 3 or 4 months of age, but in the prefrontal cortex, high rates of synaptogenesis continue until about 6 years of age ([Huttenlocher & Dabholkar, 1997](#)).

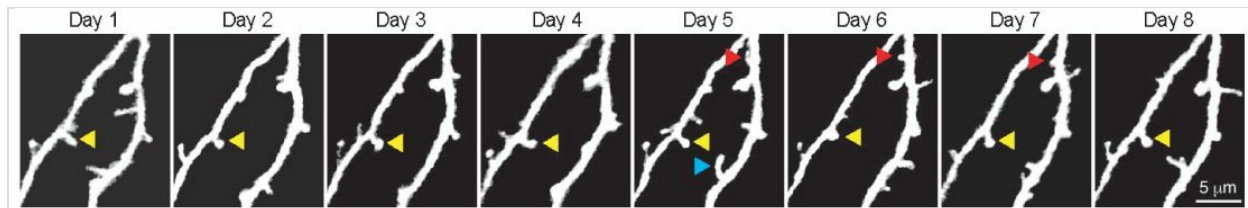
synaptogenesis

Creation of new synapses.

After this peak in synaptogenesis, the number of synapses begins to decline as the brain begins to prune unnecessary or incorrect connections. Just like neurons, synapses are subjected to a kind of Darwinian natural selection: synapses that are frequently used (and therefore presumably important to the neuron's function) are strengthened, whereas synapses that are seldom used (and therefore presumably less important) may be weakened and may die away altogether. In humans, up to 42% of all synapses in the cortex may be pruned during childhood and adolescence ([Bourgeois, 2001](#)). Despite this pruning, there are still plenty of synapses available: the adult human brain, for example, has a staggering 10^{14} synapses. That's about 1,000 times as many synapses as there are stars in the Milky Way galaxy.

Synaptogenesis continues throughout life, although not at the same furious rate as during infancy. The vast majority of synapses on cortical neurons occur on [spines](#), tiny protrusions from dendrites ([Huttenlocher & Dabholkar, 1997](#)). There may be about 100,000 spines per neuron. Throughout life, new spines periodically appear at various locations on the dendrite ([Figure 12.9](#)). If the spines are contacted by another neuron, synapses can be formed and strengthened; otherwise, the unneeded spines disappear, to be replaced in due course by new spines elsewhere on the dendrite that may prove more useful ([Trachtenberg et al., 2002](#)). Although individual

spines come and go, as shown in [Figure 12.9](#), the overall number of spines on a dendrite remains approximately constant; experience and learning determine which individual spines survive.



Karel Svoboda

FIGURE 12.9 Most synapses are located on dendritic spines This figure shows a segment of one dendrite, photographed over several consecutive days. Over a period of 8 days, this dendritic segment showed some spines that survived through the entire experiment (yellow arrowhead); some that emerged, lasted a few days, and then disappeared (red arrowhead); and some that lasted less than a single day (blue arrowhead).

Description

The images are as follows. Day 1, 2, 3, 4: A segment of dendrite with spines. A left arrow points toward a spine in the middle. The segment of dendrite with the same spine indicated by an arrowhead. On all these four days, one or two spines appear or disappear. Day 5: The segment of dendrite with the same spine indicated by an arrowhead and some new spines indicated by another arrowhead. Day 6, 7: The segment of dendrite with the same spine indicated by an arrowhead and the new lasting spine indicated by another arrowhead. Day 8: The segment of dendrite with the same spine that survived from day 1 indicated by an arrowhead.

spine

A tiny protrusion on a dendrite where synapses may form.

Sensitive Periods for Neuronal Wiring

Earlier, you read about the concept of sensitive periods, time windows during which certain types of learning are particularly efficient or effective. Sensitive periods in learning may reflect sensitive periods in neuronal development, when environmental inputs (e.g., visual stimulation) can easily alter brain organization by changing local cortical connectivity.

For example, you read that infant cats and monkeys who have one eye sewn shut during a sensitive period in infancy will be “blind” in that eye when it is opened ([Hubel & Wiesel, 1977](#)). Normally, visual stimulation activates sensory neurons in the retina of the eye that project (through several way stations) to neurons in the primary visual cortex (V1). During the first few weeks of life, this visual pathway is very active, and because “neurons that fire together wire together,” connections between neurons in this pathway are strengthened. But if one eye is

deprived of sight, there will be no activity along the pathway from that eye to V1. The inactive synapses will be weakened or eliminated, while synapses in the active pathway from the open eye will be strengthened. By the time the deprived eye is reopened, visual activity in that newly reopened eye will no longer elicit much activity in V1, and this weak activity will not be able to compete for synapses with the strong pathways from the never-closed eye ([Majewska & Sur, 2003](#)).

Although sensitive periods restrict our later learning, they are actually beneficial to the developing brain. Early in life, the brain *should* be maximally open to new experiences and be able to change accordingly. But once its basic system is set up, the brain should not change dramatically with each new experience, or it would risk overwriting critical older information. Of course, the brain remains plastic throughout life, and some reorganization can occur (e.g., the gradual changes in cortical maps that occur with extensive practice of a new motor skill, described in [Chapter 8](#)), but the basic organization must remain relatively stable from day to day. Think of your brain as a canvas on which an artist's early broad strokes define the picture being painted; later, small details can be added or changed, but the overall layout of the picture is fixed.

Brain Changes in Adolescence

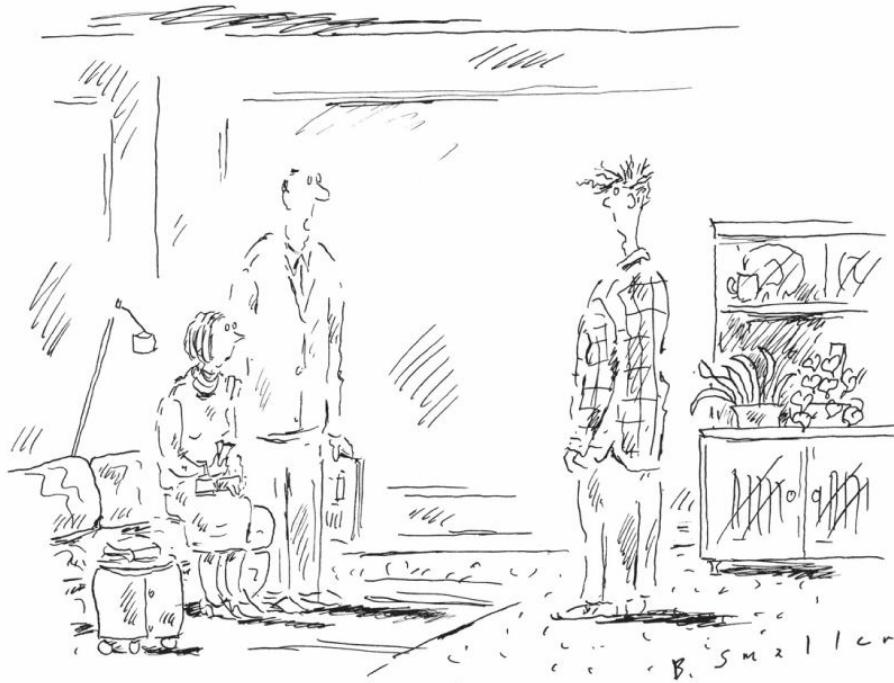
[Section 12.1](#) describes adolescence as a time of profound physical and psychological changes through which a child transitions into an adult. The psychological changes, and particularly the changes in learning and memory abilities, reflect changes in the adolescent brain.

Profound Changes in Prefrontal Cortex

Adolescence causes changes throughout the brain. Some of the most profound and obvious changes occur in the prefrontal cortex, which does not fully mature until after the teenage years. As discussed in [Chapter 9](#), the prefrontal cortex is a seat of many types of cognitive function, including working memory and also judgment and planning. During childhood, the prefrontal cortex undergoes robust synaptogenesis; but during adolescence, large numbers of these synapses are pruned until the number of synapses plateaus at the adult level ([Glantz, Gilmore, Hamer, Lieberman, & Jarksog, 2007](#)). At the same time, neurons in prefrontal cortex begin to show much more complicated branching patterns that probably allow more complex patterns of information flow ([Lambe, Krimer, & Goldman-Rakic, 2000](#)).

Another important change during development is that neuronal axons develop *myelin sheaths*, wrappings of a fatty substance called myelin that insulate the electrical signals traveling down the axon, speeding neuronal transmission. The myelin sheaths are produced by a type of *glia*, cells in the brain that provide various support functions to neurons and that are as critical to proper brain function as the neurons themselves. Myelination of neurons in the human cortex starts after birth and continues throughout the first 18 years of life. Some brain areas, such as the motor cortex and sensory cortex, are fully myelinated early, but the frontal cortex is not fully myelinated until late adolescence or early adulthood. Neurons can function before myelination is complete, but transmission will be slow and weak. This could be part of the reason that working memory is among the last brain processes to fully mature.

Yet another change in the brains of adolescent mammals is a dramatic increase in the neurotransmitter *dopamine* ([Lambe et al., 2000](#)). As [Chapter 5](#) explains, dopamine is important for learning about reward—and particularly about rewards that will happen in the future. Human teenagers experience both a redistribution of dopamine receptors around the brain and an increase in sensitivity to dopamine, which might make rewarding stimuli even more salient ([Steinberg, 2008](#)). These changes in dopamine function probably contribute to teenagers tending to be impulsive and risk-taking, and to adolescent humans (and adolescent rats and monkeys) becoming easily attracted to drugs that affect the brain's reward system ([Walker et al., 2017](#)). The peak in dopamine starts to drop off after about age 18 or 19 in humans ([Galván, 2013](#)), as teenagers begin to mature into adults who show more impulse control and more ability to act based on judgments about the long-term consequences of their behavior.



*"Young man, go to your room and stay there
until your cerebral cortex matures."*

The New Yorker Collection 2006 Barbara Smaller from
cartoonbank.com. All Rights Reserved.

If this young man's cerebral cortex is not fully mature, what learning and memory abilities might not be fully developed yet?

TEST YOUR KNOWLEDGE

Everything Grows Together ... at Different Rates

All forms of learning and memory available to adult mammals tend to be present in infants, at least in rudimentary form, but some kinds of learning mature faster than others, apparently reflecting different rates of maturation for the brain substrates on which those types of learning and memory depend. See if you can reorder the middle column of the following table to produce an accurate, if rough, timetable for when each type of learning and memory reaches maturity. Then reorganize the third column to match each type of learning and memory with a corresponding brain area. Bear in mind that this is a rough timetable only, with lots of room for individual differences as well as continued fine-tuning of abilities as the organism reaches adulthood; also remember that most kinds of learning and memory depend on more than one brain area.

([Answers appear at the end of the chapter.](#))

(Rough) Timetable for Development	Types of Learning and Memory	Critical Brain Area(s)
Present before birth (in at least some forms)	Conditioning and skill learning	Frontal cortex
Infancy to early childhood (in at	Episodic memory and	Hippocampus

least some forms)	semantic memory	
Childhood	Sensory processing and habituation	Sensory cortex
Late childhood to early adolescence	Social learning and imitation	Mirror neurons in motor cortex
Late adolescence to young adulthood	Working memory and executive function	Cerebellum and basal ganglia

Effects of Sex Hormones on Brain Organization

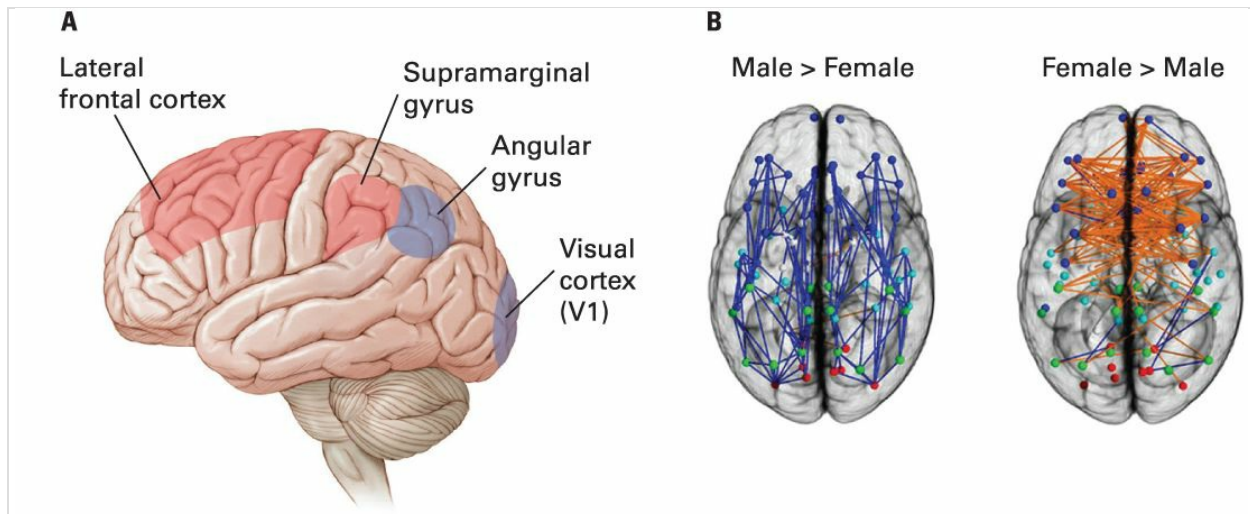
In its discussion of adolescence, [Section 12.1](#) noted several kinds of learning and memory that are different, on average, in males and females. Since many of these differences in learning and memory do not emerge until puberty, a likely explanation is that these kinds of learning and memory are strongly affected by estrogen and testosterone levels in sexually mature adults.

Not all sex hormone effects occur at puberty, though. In mammals and birds, there is a surge in testosterone near birth. This surge occurs in both sexes, although it is greater in males.

Testosterone levels then decline during the first year of life in both male and female humans, and they remain low until puberty ([Overman, Bachevalier, Schuhmann, & Ryan, 1996](#)). But during that critical first year of life, testosterone strongly influences brain development. Curiously, in the brain, “male” hormones such as testosterone are converted to estradiol (a form of the “female” hormone estrogen), so it may be the high level of estradiol in young males that actually makes their brains develop differently from female brains ([Cohen-Bendahan, van de Beck, & Berenbaum, 2005](#)).

And develop differently they do. By adulthood, men’s brains are about 100 grams heavier and contain about 4 billion more neurons, on average, than women’s brains ([Pakkenberg & Gundersen, 1997](#)). Aside from this difference in overall volume, some brain areas are proportionately larger in one sex than in the other ([Goldstein et al., 2001](#); [Goldstein et al., 2002](#); [Jacobs, Schall, & Scheibel, 1993](#)). **Figure 12.10A** shows some of these areas. One is the lateral frontal cortex, which is important for working memory; this area is usually larger in women than in men, which may help explain why women often outperform men on working-memory tasks. Other areas that are usually larger in women are the hippocampus and some language areas (e.g.,

the area labeled “supramarginal gyrus” in [Figure 12.10A](#)). This may help explain why women often outperform men on list learning, which requires both hippocampal-dependent episodic memory and language skill. Conversely, men are often better than women at navigating through space ([Astur et al., 1998](#)). Such spatial navigation may depend on brain areas that process visual and spatial information, and some of these areas tend to be larger in men than in women, such as the ones labeled “angular gyrus” and “visual cortex” in [Figure 12.10A](#).



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FIGURE 12.10 Average differences in the brains of men and women (A) Several brain areas (red) are proportionately larger in women than in men, on average, including brain areas important for working memory and language processing; women often outperform men on such tasks. Other brain areas (blue) are proportionately larger in men than women, on average, including cortical regions important for visual and spatial processing; men often outperform women on such tasks. (B) Diffusion tensor imaging (DTI) suggests that male brains typically have more intrahemispheric connectivity (blue), while female brains typically have more interhemispheric connectivity (orange).

(A) Information from [Goldstein et al., 2002](#). (B) [Sex differences in structural connectome, Madhura Ingahlalikar, et al. Proceedings of the National Academy of Sciences Jan 2014, 111 \(2\) 823–828](#).

Description

Illustration A shows the anatomy of the human brain, highlighting lateral frontal cortex, supramarginal gyrus, angular gyrus, and visual cortex (V 1). Illustration B compares male and female brains: the brain on the left labeled male is greater than female shows numerous lines connecting nodes within the left and right hemispheres while the illustration on the right labeled female is greater than male shows numerous lines connecting nodes of the left hemisphere to the right hemisphere.

Another way to examine brain structures is by using diffusion tensor imaging (DTI) to

investigate how brain areas are interconnected. One study examined the brains of nearly 1,000 participants, male and female, ranging from 8 to 22 years of age ([Ingalhalikar et al., 2014](#)). [Figure 12.10B](#) shows brain pathways that were relatively stronger in men than in women (left) or stronger in women than in men (right). The pattern is striking: most of the pathways that were stronger in men were *intrahemispheric* connections, potentially facilitating integration of information among the occipital, parietal, and frontal regions of each hemisphere. In contrast, most of the pathways that were stronger in females were *interhemispheric* connections, especially in the frontal lobes, potentially facilitating integration between the left and right hemispheres of the brain. These male–female differences did not appear in data obtained from children; they began to be apparent in data obtained from adolescents and were most clearly visible in data obtained from young adults, suggesting that these male–female differences emerge during adolescence.

In sum, sex differences in learning and memory appear to reflect the fact that male and female brains are somewhat different. Again, it is important to emphasize that “different” does not necessarily mean “better” or “worse,” and that these differences appear only *on average*; every individual’s brain is unique, and every individual’s abilities are unique. A person with strong intrahemispheric connectivity (the blue pathways in [Figure 12.10B](#)) might solve a complex problem, such as navigating to the grocery store, in a very different way than someone with strong interhemispheric connectivity (the orange pathways in [Figure 12.10B](#)). Both solutions might be equally effective, but differences in brain wiring might make men, on average, more likely to approach the problem in one way, while women might be more likely, on average, to try a different approach.

Effects of Circulating Sex Hormones on Brain Function

By the time an individual reaches sexual maturity, learning and memory abilities reflect both underlying brain organization and the effects of circulating sex hormones. These effects are not always simple to distinguish. For example, estrogen reliably stimulates neuronal growth and synaptic plasticity (LTP) in rats ([Foy et al., 1999](#); [Woolley, Weiland, McEwen, & Schwartzkroin, 1997](#)). So we might expect that women should learn better at times in their menstrual cycle when circulating estrogen levels are high than at times when estrogen levels are lower. Unfortunately, such a clear pattern has not always been found. For instance, although women normally outperform men at learning word pairs, women tested at points in their

menstrual cycle when estrogen is high do not outperform women tested when estrogen is low ([Phillips & Sherwin, 1992](#)). One reason for this confusing result may be that the fluctuations in women's estrogen levels at different points in the menstrual cycle are very small compared to the difference in estrogen levels between women and men.

On the other hand, women who are taking oral contraceptives (many of which work by raising levels of estrogen and progesterone) show faster classical conditioning than women who are not taking oral contraceptives ([Beck et al., 2008](#))—and both groups show faster conditioning than males ([Holloway, Beck, & Servatius, 2011](#)). Estrogen also affects emotional memory: naturally cycling women tend to remember more details of an emotional story than women who are using hormonal contraception ([Nielsen, Ertman, Lakhani, & Cahill, 2011](#)). Because the women in these studies were not randomly assigned to pill versus placebo groups, the studies do not rule out the possibility that other, preexisting differences between the groups account for the different levels of performance.

Another way to evaluate the effects of estrogen on learning and memory is to compare learning in cisgendered individuals (whose sense of gender corresponds with their birth sex) and in transgendered individuals. In one study, on a list-learning task, cisgendered females recalled the most words, cisgendered males recalled the fewest, and transgendered women on estrogen therapy scored intermediately—better than cisgender males but not quite as well as cisgender females ([Cohen-Kettenis, van Goozen, Doorn, & Gooren, 1998](#)). In another study, a group of transgendered women on estrogen therapy scored higher on a paired-associate task than a second group of transgendered women who had not yet started estrogen therapy ([Miles, Green, Sanders, & Hines, 1998](#)).

Whereas estrogen may improve verbal learning, there is some evidence that testosterone can improve spatial learning. For example, adult male rats normally outperform females on learning to swim to the location of an escape platform hidden just below the surface in a pool filled with cloudy water. In one study, adult male rats that were given additional testosterone performed better than untreated males ([Naghdi, Majlessi, & Bozorgmehr, 2005](#)). But other studies have found that testosterone causes no improvement or can even impair spatial memory ([Goudsmit, Van de Poll, & Swaab, 1990](#)). The results are complicated by the fact that testosterone exerts its influence on the brain mainly by affecting estrogen levels: on the one hand, testosterone inhibits

estrogen function; on the other hand, when testosterone levels are high, some testosterone may be converted to estrogen, actually increasing estrogen levels in the brain. Perhaps as researchers come to better understand the complicated relationship between estrogen and learning, they will also gain a clearer understanding of the relationship between testosterone and learning.

The Brain from Adulthood to Old Age

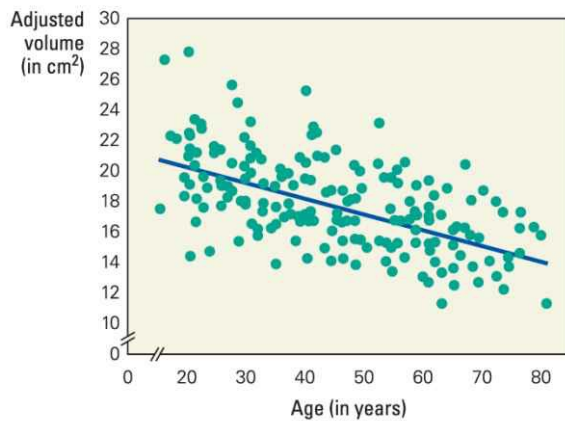
Just as the brain grows in volume during development, so does it shrink in old age. This shrinkage begins in adolescence and continues thereafter, and by age 80, the average human brain has lost about 5% of its weight. Some of this loss may reflect the early stages of degenerative disorders such as Alzheimer's disease. Some may reflect injury; a large-scale MRI study of 3,600 "healthy" individuals ages 65 to 97 years found that more than one-third had brain lesions consistent with small strokes, which had presumably gone unnoticed by the individuals themselves ([Bryan et al., 1997](#)). But even in healthy aging, where there is no known disease or injury, some brain shrinkage appears to occur.

Localized Neuron and Synapse Loss

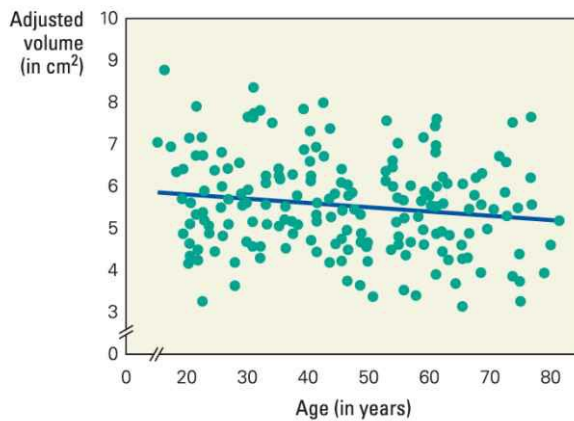
What causes brains to shrink with age? In some areas of the mammalian brain, neurons die off during the course of normal aging. For example, you know that the prefrontal cortex is important for working memory. In a young adult monkey (11 years old), the prefrontal cortex contains numerous, densely packed neurons; in an elderly monkey (25 years old), the same area of the prefrontal cortex shows nearly one-third fewer neurons ([Smith, Rapp, McKay, Roberts, & Tuszynski, 2004](#)).

Similar changes occur in humans. [Figure 12.11A](#) shows that older adults tend to have smaller volume in the lateral prefrontal cortex. This is not true for all cortical regions; thus, for example, the volume of primary visual cortex appears to decline very little with age ([Raz et al., 2004](#)). The age-related shrinkage of prefrontal cortex is consistent with the fact that working memory declines sharply with age. Tellingly, the degree of working memory impairment shown by an individual aged monkey correlates with that individual's degree of neuron loss in the prefrontal cortex ([Smith et al., 2004](#)). Another brain region where neurons are lost with age is the cerebellum ([Hall, Miller, Corsellia, 1975](#); [Woodruff-Pak & Sheffield, 1987](#)). This cell loss is consistent with the finding that classical eyeblink conditioning, which depends on the cerebellum, declines with age ([Woodruff-Pak, Logan, & Thompson, 1990](#)).

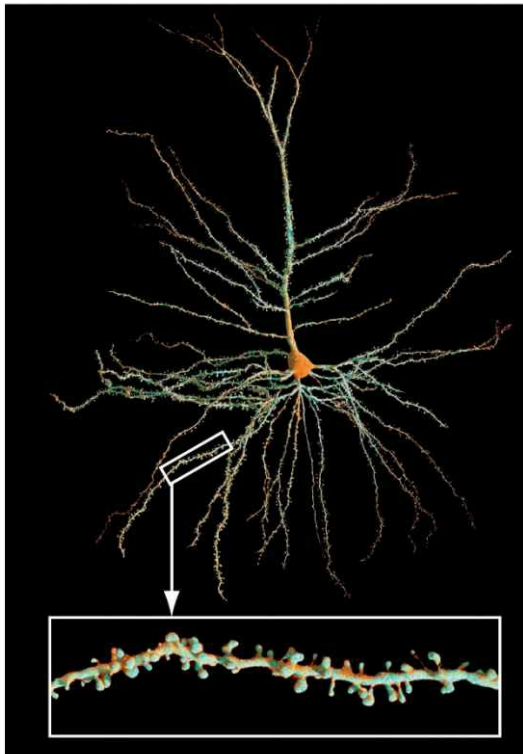
A Lateral prefrontal cortex



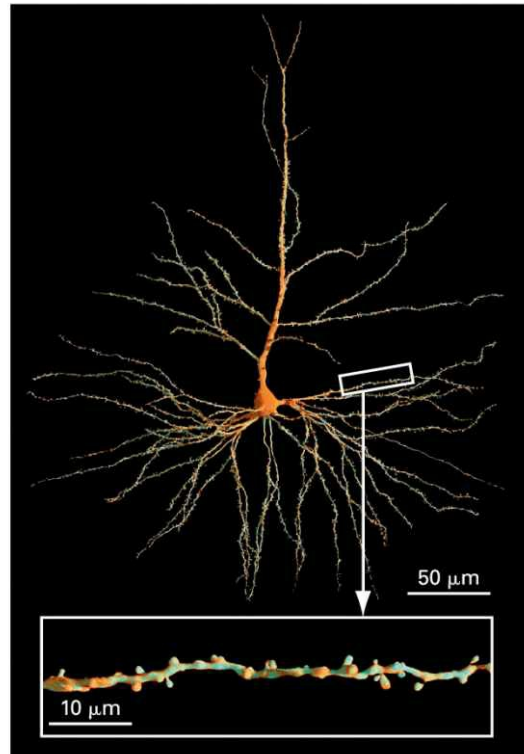
B Primary visual cortex



C Young monkey



D Aged monkey



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(C, D) John H. Morrison.

FIGURE 12.11 Aging in prefrontal cortex (A) Volume in several areas of the prefrontal cortex, including lateral prefrontal cortex, typically declines with age, shown here for humans ranging from about 20 to about 80 years in age. (B) Other cortical areas, such as primary visual cortex, do not typically show volume reductions in the course of healthy aging. (C) A representative prefrontal neuron from a young monkey; the inset shows a close-up of a segment of dendrite from this neuron, covered in spines, the tiny protrusions where synapses can form. (D) In the aged brain, the number of spines is greatly reduced.

(A, B) Research from [Raz et al., 2004](#).

Description

Scatterplots A and B have age in years on the horizontal axes and adjusted volume in centimeter square on the vertical axes.

Scatterplot A depicts the volume of lateral prefrontal cortex in humans as they age. It shows that during early adulthood or 20s, the adjusted volume of the lateral prefrontal cortex can be as high as 28-centimeter square and gradually decline to 26 or 24-centimeter square during 30s. The volume continues to decrease with increase in age and drops to 11-centimeter square by 80 years of age. It indicates a strong negative correlation.

Scatter plot B depicts the volume of primary visual cortex in humans as they age. It shows that the adjusted volume of primary visual cortex can be a maximum of 9-centimeter square during early adulthood and almost 8.5 centimeter square during the early 30s. During the 40s, it may be as low as 4.5-centimeter square or as high as 8-centimeter square. Though it shows a small decrease during the 50s, it still shows a volume above 7.5-centimeter square during old age. This indicates a weak negative correlation.

Microscopic image labeled C shows representative prefrontal neuron from a young monkey and the inset shows a close-up of a segment of dendrite from this neuron that has numerous spine-like protrusions.

Microscopic image labeled D shows prefrontal neuron from an aged monkey and the inset shows a close-up of this neuron that has fewer spine-like protrusions. A text above the inset reads, 50 mu meters, and another text inside the inset reads, 10 mu meters.

There may also be age-related decreases in the connectivity between existing neurons. For instance, neurons in the cerebral cortex of aged monkeys tend to show fewer dendrites and less complex branching than neurons of young monkeys ([Hof & Morrison, 2004](#)). Such reductions in dendritic branching imply a reduction in the ability to receive signals from other neurons ([Gallagher & Rapp, 1997](#)). Neurons with fewer dendrites also take up less space, which could be another reason some cortical areas shrink with age.

The prefrontal cortex is an area where synapse loss may be particularly marked in aging. [Figure 12.9](#) showed dendritic spines, tiny protrusions on dendrites where the majority of synapses are formed. The number of spines on prefrontal neurons decreases by about 33% overall in aged monkeys ([Figure 12.11C, D](#); [Dumitriu et al., 2010](#); [Morrison & Baxter, 2012](#)). But the loss is not universal. There are two kinds of spines: the majority (about 60 to 75%) are small, thin spines that are capable of growing and retracting and are believed to play a role in encoding new information; the minority are large, mushroom-shaped spines that tend to stay in place and are believed to be the locus of long-term memories. While aged monkeys have about 45% fewer thin spines than young monkeys, the number of mushroom spines remains approximately constant ([Dumitriu et al., 2010](#)). This could be one reason why aged individuals—as you read earlier—

often show impairments in the ability to learn new information but show little loss of old, well-established memories.

Some brain regions do not appear to lose appreciable numbers of neurons or synapses in old age, however. One such area, as noted previously, is the primary visual cortex ([Figure 12.11B](#)). Another is the hippocampus, which may shrink in overall volume but does not appear to show much age-related neuron loss in humans ([West, 1993](#)), monkeys ([Peters et al., 1996](#); [Small, Chawla, Buonocore, Rapp, & Barnes, 2004](#)), or rats ([Rapp & Gallagher, 1996](#); [Rasmussen, Schliemann, Sorensen, Zimmer, & West, 1996](#)). In fact, many researchers believe that significant reductions in the number of hippocampal neurons are warning signs of age-related disease, such as early Alzheimer's disease ([Gallagher & Rapp, 1997](#)). Similarly, it appears that the aging hippocampus does not normally show a decrease in number of synapses ([Rosenzweig & Barnes, 2003](#)). Yet, as you read in [Section 12.1](#), age does cause declines in hippocampal-dependent episodic and semantic memory formation. If not loss of hippocampal neurons, what could cause this decline?

Loss of Synaptic Stability and Consolidation

Although the *total number* of hippocampal neurons and synapses does not decline appreciably with aging, there are changes in neuronal *function*, including a reduced ability to maintain changes in synapse strength ([Barnes, Suster, Shen, & McNaughton, 1997](#); [Fan, Wheatley & Villeda, 2017](#)). Remember that synapse strength is increased by long-term potentiation (LTP), the process whereby conjoint activity in two neurons strengthens the synaptic connection between them. This is one way in which neurons encode new learning. If LTP occurs but then fades away, the new learning will be lost.

When a healthy young adult rat is placed in a new environment (e.g., a maze) for the first time, it typically moves about and explores. As described in [Chapters 3](#) and [7](#), the hippocampus contains *place cells*, neurons that fire whenever a rat is in a particular location (see [Figure 7.20](#)). While the rat explores, different place cells become active when the rat wanders into different locations in the maze. In fact, just by detecting which place cells are firing at a given moment, experimenters (and presumably the rat itself) can deduce the rat's location in the maze. If the young rat is returned to the same maze the next day, the place cells will tend to fire at the same locations as the day before. This consistency of spatial encoding helps the rat form a reliable

mental map of its environment. Obviously, the ability to learn and recognize the environment is a key part of learning and remembering how to navigate that environment.

When an older rat is first placed in the maze, it too will explore, and it too has place cells that form a map of the environment. But if the older rat is returned to the maze again tomorrow, those place cells won't always fire in the same locations as before. In effect, the place fields "shift" from day to day ([Barnes et al., 1997](#)). Apparently, both young and old rats learn about the environment and develop spatial maps of that environment, but the old rats lose this information much faster than the young rats do. Later experiments showed that LTP in an old rat's hippocampal neurons is unstable and fades after just a few hours ([Barnes, 2003](#)). This instability of hippocampal LTP in aged rats could contribute to age-related deficits in spatial learning as well as in other kinds of learning and memory that depend on the hippocampus, such as episodic memory, which requires remembering the context in which an event occurred.

There is some evidence that in humans, too, age-related declines in episodic memory and semantic memory may be due to reduction in the ability of the hippocampus to encode new information. For example, [Chapter 7](#) discusses the subsequent memory paradigm, in which researchers record fMRI activity while participants study new information such as words or pictures; later, participants are tested with a second set of items and asked whether each is new or familiar. Typically, young adults show more activity in the medial temporal lobe (including the hippocampus) and frontal lobe while studying items that will later be successfully recalled than while studying items that will later be forgotten. Older adults show the same basic pattern, but the degree of difference in hippocampal and prefrontal activation for subsequently remembered versus subsequently forgotten items is much reduced ([Daselaar, Fleck, Dobbins, Madden, & Cabeza, 2006](#); [Dennis & Cabeza, 2011](#); [Dennis et al., 2008](#)). Together, the results from animals and humans are generally consistent with the idea that age-related declines in new learning result from a general reduction in the ability to encode new information in the hippocampus and prefrontal cortex.

Why should synaptic plasticity change with age? One possible factor involves epigenetics. As you read earlier in the chapter, epigenetic processes activate and inactivate genes that trigger synaptic changes and other processes to stabilize new memory. Major mechanisms of epigenetic control, such as methylation, change with age, although the reasons are not yet clear ([Azpurua &](#)

[Eaton, 2015](#)). This would also possibly explain the wide individual differences in healthy aging: since no two individuals experience precisely the same life events, epigenetic changes vary widely—resulting in different rates of cognitive decline.

Another important factor may be sleep. As [Chapter 7](#) describes, slow-wave sleep (SWS) is thought to be important for declarative memory consolidation because it allows the hippocampus to “replay” information acquired during the day and thus reactivate and strengthen corresponding memory traces in cortex (as illustrated in [Figure 7.20](#)). Older adults typically sleep fewer hours per night than younger sleepers, and they spend proportionately more of that sleep time in light sleep than in REM and SWS ([Ohayon, Carskadon, Guilleminault, & Vitiello, 2004](#)). In addition to experiencing less SWS, older individuals may also have disruptions in brain activity during SWS. One study found that the “memory replay” observed in the hippocampus of young rats during SWS was impaired in aged rats ([Gerrard, Burke, McNaughton, & Barnes, 2008](#)). In humans, although a good night’s sleep often promotes memory for the prior day’s learning in young adults (as shown in [Figure 7.21](#)), older adults typically receive no memory boost from sleep ([Scullin, 2013](#)). Thus, reduced and disrupted SWS may be another factor impairing the ability of the aged hippocampus to encode and consolidate new memories.

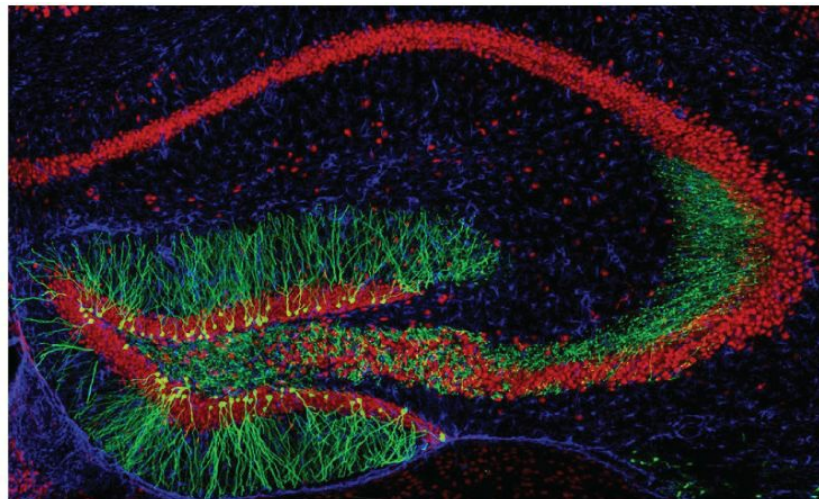
Adult Neurogenesis: New Neurons for Old Brains?

Remember the male white-crowned sparrows, who learn their song during a sensitive period early in life? In contrast, canaries are “lifelong” learners who can alter their song from year to year. Canary song is especially important during the spring, when males sing to defend their territory and to attract mates. This seasonal variation in singing is mirrored in the canaries’ brains. For example, [Chapter 11](#) describes the high vocal center (HVC), which is a brain area important for song production (see [Figure 11.10](#)). In canaries, the HVC doubles in volume during the spring relative to its size in the fall ([Ball & Hulse, 1998](#)).

To investigate this seasonal variation, Fernando Nottebohm and his colleagues injected radioactive *thymidine* into adult canaries. Thymidine is a compound that is taken up by cells undergoing mitotic division; it therefore can be used as a marker for newly born cells. The researchers found traces of thymidine in the birds’ HVC as well as in the forebrain ([Goldman & Nottebohm, 1983](#)), suggesting the presence of new neurons. Nottebohm and his colleagues demonstrated that these newly generated cells developed into functional neurons, making

connections with existing neurons and generally appearing anatomically and physiologically indistinguishable from older cells ([Burd & Nottebohm, 1985](#); [Paton & Nottebohm, 1984](#)). Thus, birds appear to have the capacity for *adult neurogenesis*, the ability to produce functional neurons throughout the lifespan.

A decade later, adult neurogenesis was also demonstrated in the brains of mice and rats, particularly in the *dentate gyrus*, which is a subregion of the hippocampus, and in the *olfactory bulb*, a brain area important in early processing of odor stimuli ([Kuhn, Dickinson-Anson, & Gage, 1996](#)). This suggested that the brain had a constantly renewed pool of “fresh” neurons available to encode new information ([Becker, 2005](#); [Kempermann, Wiskott, & Gage, 2004](#); [Wiskott, Rasch, & Kempermann, 2006](#)). Consistent with this idea, rats treated with low-dose radiation, which inhibits neurogenesis in the hippocampus, are impaired at new hippocampal-dependent learning ([Winocur, Wojtowicz, Sekeres, Snyder, & Wang, 2006](#)). On the other hand, rats in the process of new hippocampal-dependent learning tend to show higher rates of neurogenesis—suggesting that new neurons are being created on demand to encode the new information ([Epp, Spritzer, & Galea, 2007](#)). Furthermore, both physical exercise and environmental enrichment increase neurogenesis in rodents, which might be why, as [Chapter 2](#) notes, physical exercise and environmental enrichment can improve learning and memory.



Lab of FH Gage at the Salk Institute

Adult neurogenesis in the rodent brain This photo shows a cross section through the hippocampus in a mouse brain. The cell bodies of adult neurons appear red. New neurons appear in green, with their cell bodies scattered among the adult neurons in a subregion of the hippocampus called the dentate gyrus and their dendrites extending outward. Why might it be useful to have a pool of “new” neurons in the hippocampus that is constantly replenished throughout life?

Description

New neurons have their cell bodies scattered among the adult neurons in a sub-region of the hippocampus called the dentate gyrus and their dendrites extended outward.

But what about the brains of humans and other primates? In the 1990s, Swedish neuroscientist Peter Eriksson injected human cancer patients with a synthetic form of thymidine called *BrdU* (short for *bromodeoxyuridine*) in an attempt to quantify the progress of their disease by tagging proliferating cancer cells ([Eriksson et al., 1998](#)). Unexpectedly, the BrdU labeled not only cancer cells but also neurons in the basal ganglia and in the hippocampus, suggesting that these brain areas contained neurons that had been born in the past few weeks. Around the same time, Elizabeth Gould and colleagues used BrdU to tag what appeared to be newborn neurons in the cortices of adult monkeys ([Gould, Reeves, Graziano, & Gross, 1999](#)). One estimate suggested that in the adult macaque monkey, a few thousand new neurons might be born daily in the hippocampus alone ([Gould & Gross, 2000](#)). At least some of the new neurons in the hippocampus appeared to become functional in the sense of forming viable connections with other neurons ([Toni et al., 2008](#); [Vivar et al., 2012](#)).

But other researchers were not so sure. Some challenged the methodology of these studies ([Korr & Schmitz, 1999](#); [Nowakowski & Hayes, 2000](#); [Rakic, 2002](#)), arguing that the studies which appeared to demonstrate adult neurogenesis in humans and monkeys may have used methodologies that can label mature neurons and non-neuronal (glial) cells as well as new neurons, creating a false impression that newborn neurons abounded ([Rakic, 2002](#); [Sorrells et al., 2018](#)). Furthermore, the vast majority of newly born neurons appeared to die off within a few weeks after formation ([Gould & Gross, 2000](#)), suggesting that most new cells did not survive long enough to make a meaningful contribution to brain function.

The controversy deepened in early 2018. Two widely publicized studies examined postmortem samples obtained from humans ranging in age from prenatal through adult and into old age ([Cipriani et al., 2018](#); [Sorrells et al., 2018](#)); both studies found high levels of newborn neurons in the hippocampus of fetal brains but lower levels in infant brains, with new neurons becoming increasingly rare in the brains of children aged 7 to 13 and completely absent in the hippocampus of adults aged 18 to 77. These results were consistent with an earlier study that similarly found evidence of new neurons in the brains of infants but not in the brains of individuals aged 3 to 59

years ([Dennis, Suh, Rodriguez, Kril, & Sutherland, 2016](#)). Months later, however, another high-profile paper argued that, with state-of-the-art tissue-processing methods, thousands of immature neurons could indeed be observed in the adult human hippocampus, even in extreme old age ([Moreno-Jiménez et al., 2019](#)).

The controversy continues regarding whether adult neurogenesis, as reliably observed in birds and rodents, routinely occurs in the brains of primates, including humans—and, if so, whether those new neurons survive in numbers sufficient to affect brain function.

Interim Summary

- Both genes and environmental experience influence an individual's learning and memory abilities.
- Epigenetic processes, which determine whether and when specific genes are activated, play an important role in activating and inactivating genes to cause changes to neurons and synapses during memory encoding and consolidation.
- In the developing brain, neurons and synapses are first overproduced and then weeded out in a process similar to Darwinian natural selection: those that make functional connections (and presumably encode important information) survive, while others die off. Growth and pruning occur at different rates in different brain areas, perhaps helping to explain why some learning and memory abilities mature faster than others.
- Sensitive periods in learning reflect sensitive periods in neuronal development, when specific kinds of environmental input can quickly and easily alter brain organization.
- Reliable sex differences exist between males and females in several types of learning and memory, although these are differences in average performance in each group and may not accurately describe any individual male or female. Sex differences in behavior may reflect the effects of sex hormones on the developing brain as well as on the activity of the adult brain.
- In the aging brain, some areas lose appreciable numbers of neurons and synapses. In the aging hippocampus, neuronal plasticity (LTP) may become less stable, meaning that new learning may not survive for long. Age-related reductions in slow-wave sleep (SWS) may also impair the ability to consolidate declarative memory.
- Adult neurogenesis continues throughout the lifespan in birds and rodents. The extent of adult neurogenesis in humans is currently unclear.

12.3 Clinical Perspectives

This section discusses two of the many ways learning and memory can be impaired at different points in the lifespan. Down syndrome strikes individuals at the beginning of their life and causes a lifelong impairment in learning ability and other aspects of intellectual function. At the other end of the lifespan, Alzheimer's disease afflicts mainly elderly individuals, resulting in a gradual loss of memory and an intellectual decline. As different as these two syndromes may appear, it turns out that there is an intriguing connection between them.

Down Syndrome

Down syndrome is a congenital disorder that causes mild-to-moderate intellectual disability. Down syndrome occurs in about 1 of every 700 live births ([National Down Syndrome Society, 2017](#)). It affects males and females equally and occurs in all ethnic groups.



Balazs Mohai/EPA/Newscom

Individuals with Down syndrome have a characteristic facial profile, as well as delayed physical growth and mild to

moderate intellectual disability. The life expectancy for those with Down syndrome, which was once only about 25 years, has greatly increased in the past few decades, largely due to better medical care and treatment. Given what you know about the effects of environmental enrichment, how might changes in societal treatment of people with intellectual disabilities also play a role in helping individuals with Down syndrome live happier, more productive lives?

Down syndrome

A congenital disorder that causes mild-to-moderate intellectual disability and is usually due to trisomy 21.

Children with Down syndrome show slowed development of speech and language; adults with Down syndrome often have mental abilities similar to a typically developing 8- or 9-year-old, although there is wide variation. In addition, most individuals with Down syndrome have physical growth delays and a characteristic facial profile, which can include a flattened nose and folds of skin over the inner corners of the eyes. Often, there are also severe medical complications; for example, about 40% of all babies born with Down syndrome have congenital heart defects. As recently as 1980, the average life expectancy for individuals with Down syndrome was about 25 years; but largely due to better medical care and treatment (particularly for heart problems), the average lifespan has since risen to about 60 years ([National Down Syndrome Society, 2017](#)). Individuals with Down syndrome must balance the desire for adult independence, possibly including marriage and employment, with what is in many cases an ongoing need for support and care from family and community.

Down syndrome was first described by English physician John Langton Down in the mid-1800s ([Down, 1866](#)), but its genetic basis was not discovered until a century later ([Neri & Opitz, 2009](#)). As you read in [Section 12.2](#), healthy human beings carry 23 pairs of chromosomes in almost every cell of the body, with one member of each pair contributed by each parent. Down syndrome is caused by having a third copy of the genes on chromosome 21. Usually, this happens when the baby inherits an extra copy of chromosome 21, a condition called [trisomy 21](#). In most cases, the extra copy comes from the mother, a risk that rises dramatically with maternal age. The likelihood of giving birth to a child with Down syndrome is approximately 1 in 1,500 for a 20-year-old mother, but it is 1 in 1,000 for a 30-year-old, and more than 1 in 50 for a 50-year-old.

trisomy 21

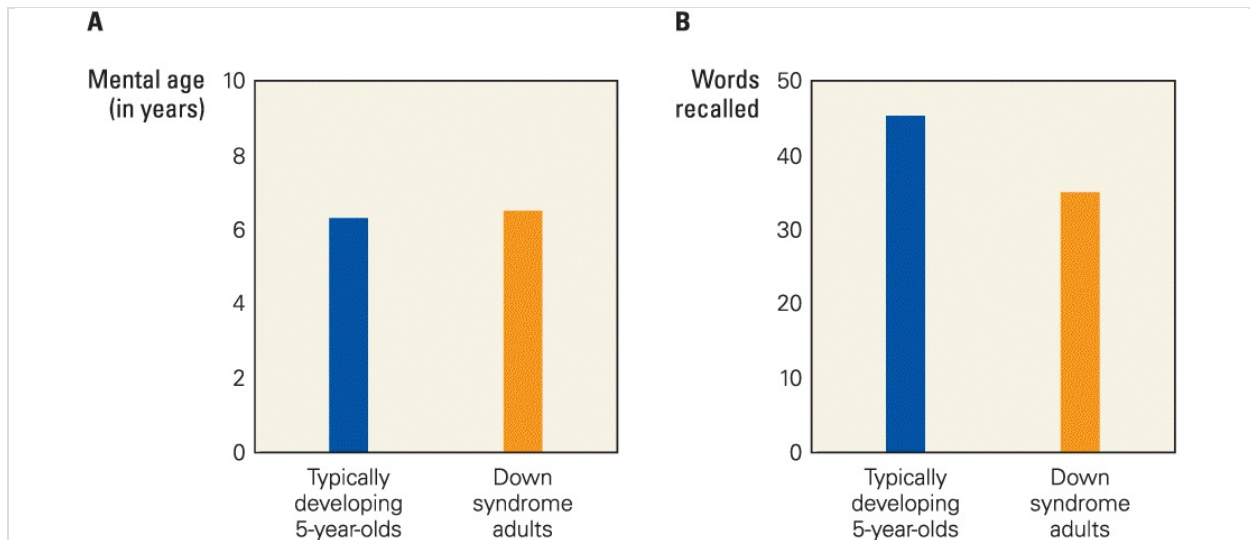
A condition in which an embryo inherits three (rather than two) copies of chromosome 21, resulting in Down syndrome.

Brain Abnormalities and Memory Impairments in Down Syndrome

Chromosome 21 contains several hundred genes ([Wiseman, Alford, Tybulewicz, & Fisher, 2009](#)). At present, researchers do not know exactly which gene or genes cause the intellectual disability in Down syndrome; several candidate genes have been identified, and any or all of them may contribute ([Crnic & Pennington, 2000](#)). We do know that the brains of individuals with Down syndrome appear normal at birth, but by 6 months of age, the brains are visibly smaller than those of other same-age children ([Nadel, 2003](#)).

By adolescence, several key brain areas are particularly small in individuals with Down syndrome, including the hippocampus, frontal cortex, and cerebellum ([Wiseman et al., 2009](#)). As a result, we might expect to see particularly severe impairments in memory tasks that depend on these brain areas. To some extent, this seems to be the case ([Godfrey & Lee, 2018](#)).

An example comes from a study involving a group of young adults with Down syndrome ([Vicari, Bellucci, & Carlesimo, 2000](#)). On standard intelligence tests, these particular individuals were found to have an intelligence level slightly above that of a group of 5-year-old typically developing children ([Figure 12.12A](#)), but on a hippocampal-dependent memory task—recalling words from a studied list—the adults with Down syndrome performed significantly worse than the 5-year-olds ([Figure 12.12B](#)). By the same token, children with Down syndrome tend to be greatly impaired at recalling recent episodic events, such as what they had for breakfast or what time they went to bed last night ([Pennington, Moon, Edgin, Stedron, & Nadel, 2003](#)). Thus, although Down syndrome involves general intellectual disability, the impairments in Down syndrome seem particularly profound for memory abilities that depend on the hippocampus ([Nadel, 1999, 2003](#)).



Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

FIGURE 12.12 Impairments in Down syndrome are often particularly severe for hippocampal-dependent learning (A) A group of young adults (average age 21 years) with Down syndrome had an intelligence level (“mental age”) comparable to that of a group of typically developing 5-year-old children. (B) However, on hippocampal-dependent tests, such as recalling words from a studied list, the adults with Down syndrome performed much worse than the 5-year-olds.

Data from [Vicari et al., 2000](#).

Description

Graph A shows the Typically developing 5-year-olds and Down syndrome adults on the horizontal axis and mental age in years from 0 to 10 on the vertical axis. The mental age of Down Syndrome adults is 6, which is also the mental age of typically developing 5-year-olds.

Graph B shows the Typically developing 5-year-olds and Down syndrome adults on the horizontal axis, and Words recalled along the vertical axis from 0 to 50. The developing 5-year-olds recalled about 45 words, and adults with Down Syndrome could recall approximately 35 words.

Animal Models of Down Syndrome

Since we know that trisomy 21 causes Down syndrome in humans, researchers have worked to develop animal models with the same genetic abnormality. In mice, for instance, a large region of chromosome 16 appears to serve the same functions as a region on human chromosome 21. Mice born with trisomy 16 (a third copy of their 16th chromosome) generally do not survive birth. (Down syndrome is also a common cause of miscarriage in humans.) So researchers have bred mice with trisomy of only a segment of chromosome 16 (a condition called *segmental trisomy*). A widely studied example is the Ts65Dn mouse strain ([Davisson et al., 1993](#)).

Ts65Dn mice can be contrasted with littermates that share most of the same genes and environment but that do not have the trisomy. Compared to their littermates, Ts65Dn mice have irregularities in the hippocampus and cortex, just like humans with Down syndrome ([Belichenko et al., 2004](#)). The Ts65Dn mice also show memory deficits reminiscent of the memory deficits in humans with Down syndrome. For example, Ts65Dn mice show impairments on hippocampal-dependent tasks, such as remembering the goal location in a maze ([Escorihuela et al., 1995](#)).

Ts65Dn mice also have excessive neuronal inhibition in the hippocampus and cortex, which may contribute to their learning and memory deficits. Two genes that have a role in the balance between excitation and inhibition of neurons in the human brain are the *Olig1* and *Olig2* genes on chromosome 21; three rather than two copies of both these genes are present in humans with Down syndrome and also in Ts65Dn mice, perhaps causing over-inhibition in the hippocampus. When given drugs that reduce neuronal inhibition (by interfering with the inhibitory neurotransmitter GABA), Ts65Dn mice show significant improvement of hippocampal-dependent learning and memory ([Fernandez et al., 2007](#)). When the number of gene copies is re-normalized (by breeding Ts65Dn females with males that have only one copy each of the *Olig1* and *Olig2* genes), hippocampal inhibition is restored to normal levels in the offspring ([Chakrabarti et al., 2010](#)). Thus, the mouse models suggest that these two genes may play a key role in the learning and memory deficits in humans with Down syndrome.

Mouse models have also been used to study how behavioral therapy can improve cognitive function in Down syndrome. One intriguing experiment has shown that being housed in an enriched environment improves spatial memory in Ts65Dn females (though they never do quite as well as control mice). But environmental enrichment actually *exacerbates* the spatial memory impairment in Ts65Dn males ([Martínez-Cué et al., 2002](#)). This difference is curious, but the overall result is encouraging because it suggests that appropriately enriched postnatal experiences might help remediate the memory deficits associated with Down syndrome in humans.

Alzheimer's Disease

In 1901, German neuropathologist Alois Alzheimer examined a 51-year-old woman named Auguste Deter who could no longer remember her husband's name or even her own last name. Her husband reported her symptoms to include memory loss, delusions, and emotional

instability. Auguste was experiencing a type of **dementia**, a progressive cognitive decline usually due to accumulating brain pathology. When Auguste died 5 years later, Alzheimer examined her brain and found curious abnormalities, including neurons that were bunched up like knotted ropes. Thinking Auguste was a unique case, Alzheimer published a report on his findings ([Alzheimer, 1907/1987](#)). The condition, which became known as **Alzheimer's disease**, is a form of dementia characterized by accumulation of abnormalities known as “plaques” and “tangles” in the brain (more about these later). At the time of Alzheimer's initial report, average life expectancy was only about 50 years, so conditions that selectively affected older people appeared to be rare. Now we know that Alzheimer's disease is not rare at all.

dementia

Progressive cognitive decline, usually due to accumulating brain pathology.

Alzheimer's disease

A form of dementia due to accumulating brain pathology (specifically, amyloid plaques and neurofibrillary tangles).

Alzheimer's disease afflicts an estimated 5.7 million people in the United States alone, including nearly one-third of people aged 85 or over ([Alzheimer's Association, 2018](#)). The disease appears to have similar incidence throughout the world, although it seems to affect women more often than men ([Alzheimer's Association, 2018](#)), possibly because women have longer life expectancy (i.e., men may be more likely to die from other causes before developing dementia). There is currently no cure. Famous victims include film stars Charlton Heston and Rita Hayworth, civil rights activist Rosa Parks, country music legend Glen Campbell, and Ronald Reagan, who at age 89 no longer remembered having once been president of the United States.

Progressive Memory Loss and Cognitive Deterioration

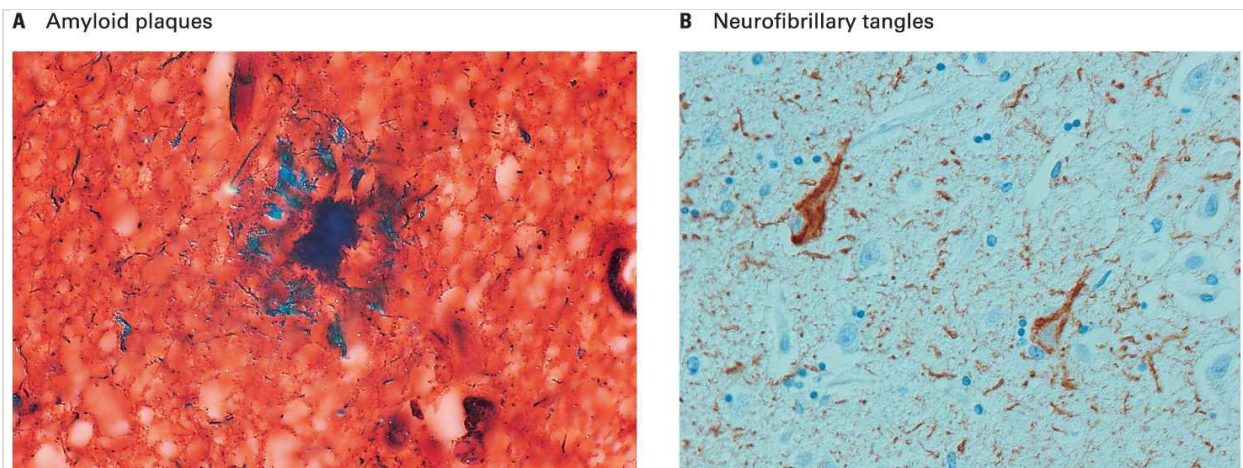
For many patients, the earliest symptoms of Alzheimer's disease are episodic memory disruptions, such as failures to remember recent conversations or visitors ([Collie & Maruff, 2000](#)). As the disease progresses, patients show marked declines in semantic memory, forgetting the names of acquaintances or the layout of familiar locations such as the neighborhood supermarket. Patients may ask the same question over and over again, forgetting not only the answer but also the fact that they have asked the question before.

Among the memory processes that survive the longest in Alzheimer's patients are conditioning and skill memory. For example, many patients can still execute well-learned skills such as making tea ([Rusted, Ratner, & Sheppard, 1995](#)) and can acquire new motor skills such as mirror

tracing ([Gabrieli, Corkin, Mickel, & Growdon, 1993](#)) and rotary pursuit ([Heindel, Salmon, Shults, Walicke, & Butters, 1989](#)), even when they cannot remember verbal information for more than a few minutes. But by the late stages of the disease, memory fails completely, and other cognitive systems begin to fail, too. Patients with late-stage Alzheimer's may display personality changes, disorientation, loss of judgment, confusion, loss of speech, and eventually an inability to perform daily activities such as bathing, dressing, and eating. At this point, patients may require round-the-clock supervision and professional care.

Plaques and Tangles in the Brain

Technically, Alzheimer's disease is defined not by its behavioral symptoms, such as memory loss, but rather by the presence of two kinds of pathology in the brain: amyloid plaques and neurofibrillary tangles. **Amyloid plaques** are deposits of *beta-amyloid*, which is a by-product of a common protein called amyloid precursor protein, or APP ([Figure 12.13A](#)). Amyloid plaques accumulate in the brain of a patient with Alzheimer's disease, and some researchers believe the plaques are toxic to nearby neurons. In 2000, an experimental vaccine was developed that could clear out amyloid plaques in human brains, but it did not have any significant effect on the cognitive symptoms in Alzheimer's patients, so it was never approved for use as a treatment ([Holmes et al., 2008](#)). Other researchers have suggested that the amyloid plaques are not the true culprit in Alzheimer's disease but rather an innocent by-product—or even evidence of the brain's attempt to isolate and protect against damaging types of beta-amyloid ([Gandy et al., 2010](#); [Nikolaev, McLaughlin, O'Leary, & Tessier-Lavigne, 2009](#)).



A: Dr. Cecil H. Fox/Science Source; B: Dennis Dickson

FIGURE 12.13 The hallmarks of Alzheimer's disease in the brain (A) An amyloid plaque (the dark spot in the center of the image), surrounded by a residue of degenerating cells. (B) Neurofibrillary tangles (seen as dark brown areas) within

neurons.

amyloid plaque

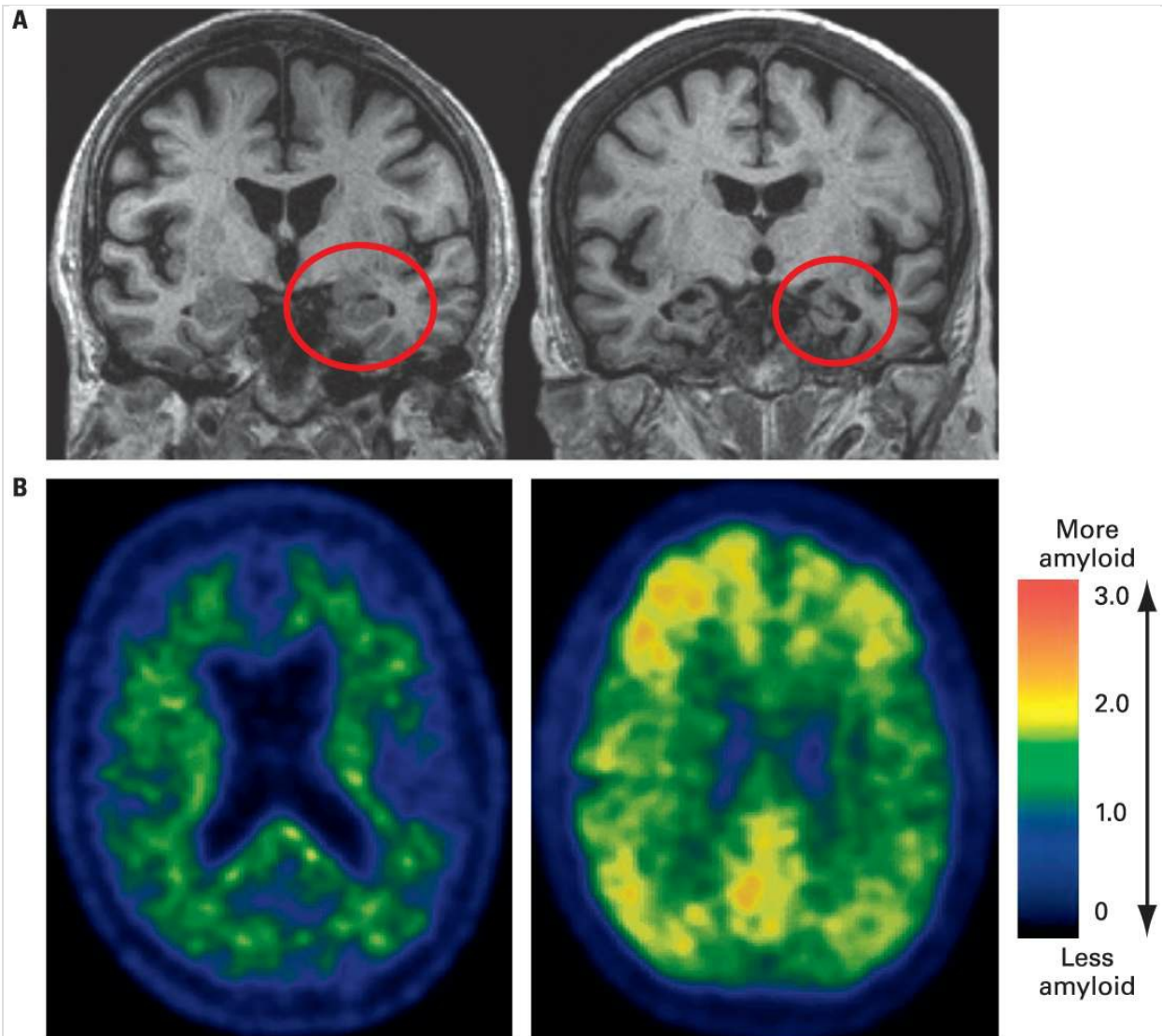
A deposit of beta-amyloid protein; these deposits accumulate in the brains of patients with Alzheimer's disease.

The second hallmark of Alzheimer's disease in the brain is the presence of **neurofibrillary tangles**, the “knotted ropes” Alois Alzheimer described in his original report. Neurofibrillary tangles are the collapsed wreckage of abnormal tau proteins that normally function as scaffolding to hold a neuron in place and that help ferry nutrients around the cell ([Figure 12.13B](#)). In Alzheimer's disease, tau molecules undergo changes and begin to clump together, forming tangles and causing the neuron to disintegrate. Some researchers believe that tau, rather than the amyloid, holds the real key to understanding Alzheimer's disease ([Johnson & Bailey, 2002](#)). Still others believe the main culprit is the immune system, which, while aggressively clearing up the residue of dead and dying cells, may also damage nearby healthy neurons ([Regen, Hellmann-Regen, Costantini, & Reale, 2017](#)).

neurofibrillary tangle

An aggregation of collapsed remains of the tau proteins that normally hold a neuron in place and help transport nutrients around the cell; such tangles occur in the brains of patients with Alzheimer's disease.

As plaques and tangles accumulate in the brain, synapse loss and neuron death occur on a large scale. Amyloid plaques tend to be fairly evenly distributed throughout the cerebral cortex, and the hippocampus is relatively spared. In contrast, neurofibrillary tangles accumulate first in the hippocampus and nearby areas. The hippocampus and nearby brain areas of patients with early Alzheimer's disease are smaller than normal, as can be seen on structural MRI ([Figure 12.14A](#); [de Leon, George, Stylopoulos, Smith, & Miller, 1989](#); [Risacher et al., 2009](#)). This pattern of hippocampal damage with relative sparing of frontal lobes in early Alzheimer's is the opposite of “healthy” aging, which, as you read earlier, tends to involve shrinkage of the cerebral cortex with relatively little loss of neurons or synapses in the hippocampus ([Braak & Braak, 1997](#); [Price & Morris, 1999](#)). In fact, among cognitively normal elderly individuals, volume reductions in the hippocampus and nearby medial temporal structures (e.g., the entorhinal cortex) indicate high risk that cognitive decline and Alzheimer's will develop over the next few years ([Apostolova et al., 2010](#); [Duara et al., 2008](#); [Risacher et al., 2009](#)).



(A) Scheltens, P. "Imaging in Alzheimer's disease." *Dialogues in Clinical Neuroscience* 11(2009): 191–199. Figure 2. (B) Courtesy of Clifford Jack at Mayo Clinic through NIH funds U01 AG06786 and R01 AG11378

FIGURE 12.14 Brain changes in early Alzheimer's disease (A) Structural MRI images from the brains of two 75-year-old individuals, one a healthy control (left) and one a patient with Alzheimer's (right). Views are coronal (as if the patients were standing upright and facing the camera); red circles have been added to indicate the location of the hippocampus in each person's left hemisphere. The hippocampus (in both hemispheres) is noticeably smaller in the person with Alzheimer's. (B) PET images from the brains of two cognitively healthy elderly individuals, injected with a contrast agent that binds to beta-amyloid. Views are horizontal (as if the people were lying on their backs, with noses in the air). Areas with high levels of amyloid appear as yellow and orange, low levels as green and blue. The individual on the left shows little or no evidence of amyloid, whereas the individual on the right shows evidence of amyloid that may indicate presymptomatic Alzheimer's disease.

Description

The structural M R I labeled A shows the brain of two 75-year old individuals, one a healthy control and one with Alzheimer's. Circles indicate the location of the hippocampus in each person's left hemisphere; the person with Alzheimer's has a smaller hippocampus. The P E T images labeled B show the levels of amyloid in two individuals which are to be read according to the color scale on the right: the individual on the left shows very little or hardly any trace of amyloid, while the individual on the right shows amyloid levels pointing to presymptomatic Alzheimer's disease. The scale reads, 0, 1.0, 2.0, and 3.0. 0 indicates less amyloid, and 3.0 indicates more amyloid.

In the past few years, it has become widely accepted that brain changes such as accumulation of amyloid plaques and neurofibrillary tangles, as well as shrinkage of brain areas such as the hippocampus, begin during an early and prolonged “presymptomatic” phase of Alzheimer's ([Frigerio & De Strooper, 2016](#)). Researchers posit that the underlying pathology is a slow process that occurs over decades and that only when the pathology accumulates to a tipping point do cognitive and clinical symptoms appear. The presymptomatic phase is followed by a second phase, sometimes called *mild cognitive impairment (MCI)*, in which mild cognitive symptoms become evident; these are usually deficits in episodic memory that do not yet meet criteria for dementia. All the while, pathology continues to accumulate beneath the surface. Some people will never go beyond these phases. But in principle, if these individuals live long enough, the pathology will eventually be so severe that serious cognitive impairments will begin to emerge.

The idea of a long presymptomatic phase raises the possibility that the disease could be recognized and even treated before serious clinical symptoms emerge. An important research area is the effort to develop reliable, inexpensive, and safe methods to detect these underlying brain changes. Some approaches center on measuring levels of beta-amyloid—for instance, by injecting a radioactive compound that binds to amyloid plaques, allowing the plaques to be visualized on PET scans. [Figure 12.14B](#) shows examples of such PET scans of the brains of two elderly individuals. Although both these people appeared to be cognitively normal, one brain shows high levels of beta-amyloid, suggesting an early stage of Alzheimer's pathology which over the next few years will begin to produce cognitive impairments ([Jack et al., 2010](#)). Similar methods are available to detect evidence of abnormal tau protein. These techniques for detecting evidence of abnormal levels of amyloid and tau provide measures that correlate well with degree of Alzheimer's symptoms and can help predict risk for future cognitive decline in elderly individuals who do not yet show symptoms. However, they are not currently considered sufficient to provide a definitive diagnosis of Alzheimer's (although they may be used to rule out other possible causes of dementia).

Another approach to detecting Alzheimer's disease at an early stage has been to focus on recognizing behavioral symptoms. [Chapter 6](#) discusses the role of the hippocampus and associated hippocampal region structures in such tasks as acquired equivalence that require the ability to generalize when familiar information is presented in a new way. Individuals with atrophy of the hippocampus might be selectively impaired at such generalization, possibly exhibiting subtle deficits well before the onset of clinical symptoms of Alzheimer's disease. Some early evidence suggests that this is so. Catherine Myers and colleagues gave a test of acquired equivalence to a group of elderly individuals who had each also undergone MRI to assess hippocampal volume. The individuals with normal hippocampal volume performed well on the test, but those with mild to moderate hippocampal atrophy (equivalent to a volume reduction of about 10 to 15%) were severely impaired at generalization ([Myers et al., 2003a](#)).

In other research, nondemented elderly participants who showed poor generalization were more likely to show symptoms of cognitive decline 2 years later ([Myers, Kluger, Golomb, Gluck, & Ferris, 2008](#)). And individuals who are carriers of genetic mutations that cause a rare early-onset form of Alzheimer's but who do not yet show symptoms of dementia exhibit impaired generalization relative to family members who do not carry the gene ([Petok et al., 2018](#)). Data such as these suggest that poor performance on tests of hippocampal-dependent generalization might serve as an early warning signal to indicate which individuals are at highest risk of developing Alzheimer's disease within the next few years.

Genetic Basis of Alzheimer's Disease

To date, several genes have been implicated in Alzheimer's disease. Most progress has been made in understanding the genetic basis of a rare, early-onset form of Alzheimer's. This early-onset Alzheimer's represents fewer than 1% of all cases of the disease, but it is especially heartbreaking because it can strike people as young as 35 to 50 years old. Mutations on three genes are associated with early-onset Alzheimer's: the *APP* gene on chromosome 21, the *presenilin-1 (PS1)* gene on chromosome 14, and the *presenilin-2 (PS2)* gene on chromosome 1. A child who inherits just one of these mutated genes from either parent (and who lives long enough) will inevitably develop Alzheimer's.

Another gene, *APOE* on chromosome 19, has been associated with the more common, late-onset form of Alzheimer's, which appears in individuals age 60 or older. *APOE* comes in several

alleles, three of which—called *E2*, *E3*, and *E4*—occur frequently in the general population. The *E3* allele is the most common and is considered the “normal” allele; about 61% of people in the United States carry two copies of *E3*, one inherited from each parent ([Alzheimer’s Association, 2018](#)). Compared to people with two copies of *E3*, people who carry one copy each of *E3* and *E4* are at 3 times higher risk of developing Alzheimer’s, and people carrying two copies of *E4* are at 15 times higher risk ([Blennow, de Leon, & Zetterberg, 2006](#)). On the other hand, having one or two copies of the *E2* allele reduces the risk of developing Alzheimer’s.

At present, it is unclear why these alleles have such effects. Some studies suggest that *E4* carriers have fewer dendritic spines in the hippocampus than *E3* carriers do, but we do not yet know whether this difference is a cause or an effect of developing Alzheimer’s disease ([Teter & Finch, 2004](#)). Another possibility is that the *APOE* gene may help control how the brain clears out beta-amyloid; some alleles (e.g., *E2*) may perform this function well, but other alleles (e.g., *E4*) may be less effective and allow plaques to build up, contributing to the brain pathology in Alzheimer’s patients.

A number of other genes have been identified as affecting an individual’s risk for late-onset Alzheimer’s disease, including the *TREM2*, *CLU*, *PICALM*, and *CR1* genes ([Jonsson et al., 2013](#); [Jun et al., 2010](#); [Seshadri et al., 2010](#)). But genes alone do not tell the whole story. For example, some individuals carrying two copies of the “good” *APOE E2* allele still develop late-onset Alzheimer’s, and some individuals carrying two copies of the “bad” *APOE E4* allele never do. It seems likely that these genes merely *predispose* a person to develop Alzheimer’s, meaning that additional environmental factors (e.g., exposure to toxins, poor vascular health, brain inflammation) may be necessary to actually trigger the disease.

The good news is that if environmental factors help trigger the disease, then changing these factors might help prevent it. Right now, evidence suggests that physical exercise—even light physical activity once or twice per week—can improve cardiovascular health, increasing blood flow with its cargo of oxygen and nutrients throughout the body and brain and helping to protect against cognitive decline and Alzheimer’s disease ([Radak et al., 2010](#); [Schlosser Covell et al., 2015](#)). Physical exercise may also improve function in people already diagnosed with probable Alzheimer’s ([Rao, Chou, Bursley, Smulofsky, & Jezequel, 2014](#)). Although many studies have searched for a similar effect of mental exercise in protecting against Alzheimer’s, the evidence to

date is not as strong. (For more on this, see “[Learning and Memory in Everyday Life](#)”.)

LEARNING AND MEMORY IN EVERYDAY LIFE

Can Mental Exercise Protect Against Alzheimer’s Disease?

Many researchers have argued that high levels of cognitive activity (“mental exercise”) can help protect against Alzheimer’s disease. There is accumulating evidence that older individuals who participate in mentally challenging pastimes—reading, playing chess, or doing crossword puzzles—are less likely to experience cognitive decline than those who stick to more passive pursuits, such as watching television ([Bennett et al., 2003](#); [Wilson & Bennett, 2003](#)). Individuals with a high educational level or a wide social support network are also at lower risk of Alzheimer’s; even when such individuals do develop the disease, they can often tolerate a greater degree of brain pathology before they begin to show clinical symptoms ([Scarmeas & Stern, 2004](#)). In all these cases, the common theme is that mental activity protects against Alzheimer’s disease—leading to the common exhortation of “use it or lose it.”

One interpretation of these findings is that exercising your brain builds up a *cognitive reserve*. On average, individuals who have been cognitively active throughout life enter old age at a high level of cognitive function. If Alzheimer’s disease strikes, these individuals have further to decline before showing sufficient loss of function to interfere with daily life ([Scarmeas & Stern, 2004](#); [Stern, Albert, Tang, & Tsai, 1999](#)).

A second, not mutually exclusive, possibility is that cognitive activity actually combats Alzheimer’s disease. [Chapter 2](#) describes how *environmental enrichment* can lead to changes in the brains of young rats, including growth of new neuronal connections (as seen in [Figure 2.6](#)). Similar changes in the brains of physically and cognitively active people might help them compensate for the loss of neurons and connections that occur in early Alzheimer’s ([Nithianantharajah & Hannan, 2009](#); [Wilson & Bennett, 2003](#)).

So far, the studies linking cognitive activity with reduced Alzheimer’s risk are only observational, meaning that they cannot prove a cause–effect relationship. On the one hand, it is indeed possible that mental exercise helps prevent Alzheimer’s and that people who choose to spend their time on mentally challenging pursuits, such as crossword puzzles, are in fact protecting themselves against the risk of developing Alzheimer’s. But it is also possible that an individual in the earliest (presymptomatic) stages of Alzheimer’s is simply less competent at such mentally challenging pursuits and so is unlikely to choose to engage in them—just as someone with chronic joint pain might be unlikely to choose to engage in extreme physical sports. Viewed this way, cognitive inactivity would not be a *cause* of Alzheimer’s but rather a *symptom*.

In March 2017, an independent panel of experts conducted a systematic review of the literature and concluded that there simply is not yet solid evidence that mental exercise protects against Alzheimer’s disease ([Kane et al., 2017](#)). One reason may be that no *single* lifestyle choice determines whether we will or won’t get Alzheimer’s disease. Rather, it may be that a number of lifestyle choices—including healthy diet, regular low-level exercise, and varied mental exercise and social stimulation—all work together to help prevent or at least postpone the onset of cognitive decline.

A Connection Between Down Syndrome and Alzheimer’s Disease

Did you notice a connection between the genes implicated in Down syndrome and Alzheimer's disease? Chromosome 21, the chromosome that is tripled in Down syndrome, also contains the *APP* gene, which is implicated in one form of Alzheimer's disease. This intriguing fact has led researchers to look for connections between Down syndrome and Alzheimer's. In fact, almost all people with Down syndrome who survive beyond age 40 develop plaques and tangles in their brains—just like Alzheimer's patients ([Moncaster et al., 2010](#)). And about 50% of adults with Down syndrome show memory decline and other symptoms similar to the cognitive impairments in Alzheimer's disease ([Brugge et al., 1994](#); [Cutler, Hesten, Davies, Haxby, & Schapiro, 1985](#)). You read earlier about Ts65Dn mice as an animal model of Down syndrome. In these mice, learning and memory can be improved by drugs that reduce beta-amyloid ([Netzer et al., 2010](#)) as well as by the Alzheimer's drug memantine ([Rueda et al., 2010](#)), so there is some hope that these drugs may also improve cognition in humans with Down syndrome.

But the other 50% of adults with Down syndrome do not show age-related cognitive decline—even though they have the same brain pathology! As yet, it is not clear why this should be. But the explanation might add to our understanding of both Alzheimer's disease and Down syndrome and suggest some possible therapies to help prevent such brain pathology from causing memory decline.

Interim Summary

- Down syndrome is a condition in which babies are born with an extra copy of chromosome 21.
- Individuals with Down syndrome have cognitive impairments, including memory deficiencies, and certain brain areas, including the hippocampus, frontal cortex, and cerebellum, tend to be abnormally small.
- Alzheimer's disease is a form of dementia in which amyloid plaques and neurofibrillary tangles accumulate in the brain. Pathology accumulates slowly, over decades, before cognitive and clinical symptoms emerge.
- Memory symptoms are prominent early in Alzheimer's disease, consistent with the finding that the hippocampus and nearby medial temporal areas suffer pathology early in the disease.
- Genes that cause rare, early-onset forms of Alzheimer's disease have been identified; other genes may contribute to an individual's risk of developing the common late-onset form of the disease.

- There may be a connection between Down syndrome and Alzheimer's disease, because chromosome 21, which is tripled in Down syndrome, also contains one of the genes implicated in an early-onset form of Alzheimer's disease. Furthermore, almost all people with Down syndrome who live long enough develop Alzheimer's-like plaques and tangles in their brains.

Synthesis

At the beginning of this chapter, you met Denise, a quintessential “healthy young adult,” at the peak of physical health and also of memory ability. Aside from appreciating her own memory abilities, how might Denise apply the lessons of this chapter next time she comes home for the holidays?

First, consider Denise’s young niece Kelly. At 10 months of age, many areas of Kelly’s brain are still undergoing intensive neurogenesis, synaptogenesis, and myelination. Yet Kelly is already quite capable of habituation, conditioning, skill learning, and observational learning, and she can express this learning by action and imitation, if not yet by words. On the other hand, her episodic memories may not yet have the same richness of information about spatial and temporal context as adult memories. In addition, Kelly’s frontal lobes are not yet fully operational, which means she may have trouble juggling several pieces of information in memory at the same time. This works to the family’s advantage when Kelly cries in frustration: it is still fairly easy to distract her attention from whatever upsets her, by waving a shiny, brightly colored toy.

Denise may also be able to apply learning and memory principles to her interactions with her grandparents. Although her grandmother seems to be handling old age well, Denise can expect that there will be some deficiencies in the older woman’s working memory and in her ability to form new episodic memories, and Denise can be understanding and supportive when her grandmother misplaces something or tells a favorite story for the tenth time. But Denise should take advantage of her grandmother’s fund of semantic and episodic knowledge while it is still available, perhaps encouraging the older woman to create a family cookbook or written history to preserve these memories for future generations.

Denise’s grandfather is a more difficult case. Instead of chalking up his memory deficits to old age, Denise should encourage the family to take him for a thorough neuropsychological exam to determine whether his memory lapses might reflect Alzheimer’s disease or whether they stem from some other condition that might be treatable. If it is Alzheimer’s, then the sad fact is that Denise’s family is in for a long struggle as they watch her grandfather slowly decline and become more dependent on a caregiver’s assistance. Although there is currently no way to reverse or prevent the cognitive decline in Alzheimer’s disease, the younger members of the family can hope that by the time they become grandparents themselves, better treatments will be

available, increasing their chances of facing old age with their brains and memories intact.

Chapter 12 Review

Know Your Key Terms

[adolescence](#)

[allele](#)

[Alzheimer's disease](#)

[amyloid plaque](#)

[androgen](#)

[apoptosis](#)

[chromosome](#)

[dementia](#)

[DNA](#)

[Down syndrome](#)

[elicited imitation](#)

[epigenetics](#)

[estrogen](#)

[gene](#)

[gestational age](#)

[imprinting](#)

[neurofibrillary tangle](#)

[neurogenesis](#)

[puberty](#)

[sensitive period](#)

[spine](#)

[synaptogenesis](#)

[testosterone](#)

[trisomy 21](#)

Quiz Yourself

1. _____ refers to the process of physical change during which the body transitions to sexual maturity. ([p. 512](#))

2. A spine is a protrusion on a(n) _____ where a(n) _____ may form. (p. 527)
3. Down syndrome is a form of _____, usually caused by a genetic condition called _____, in which the embryo inherits three (rather than two) copies of a specific chromosome. (p. 539)
4. The _____ are the principal sex hormones present in adult females. The _____ are the principal sex hormones present in adult males, and the most important of these is _____. (p. 515)
5. _____ is a technique for assessing memory in infants that involves observing their ability to mimic actions they have seen earlier. (p. 506)
6. _____ is a form of dementia usually characterized by memory impairments as an early symptom. (p. 541)
7. A naturally occurring variant of a gene is called a(n) _____. (p. 521)
8. The _____ age of an embryo or fetus is the age in terms of time since conception. (p. 502)
9. Whereas _____ encode the instructions for building and maintaining an organism, _____ mechanisms determine how and when those instructions are executed. (p. 524)
10. The brains of patients with Alzheimer's disease accumulate brain pathology in the form of _____, which are deposits of beta-amyloid protein, and _____, which are collapsed remains of the proteins that normally hold a neuron in place. (p. 542)
11. _____ is the transitional stage between puberty and adulthood, corresponding roughly to the _____ years in humans. (p. 513)
12. Natural cell death, not caused by accident or disease, is called _____. (p. 527)
13. The tendency of youngsters in some species to form an attachment to the first

individual they see after birth is called _____. (p. 508)

14. _____ refers to the creation of new neurons in the brain; _____ refers to the creation of new synapses. (pp. 526–527)
15. A time window, usually early in life, during which a certain kind of learning is most effective, is known as a(n) _____ for that learning. (p. 508)
16. _____ refers to progressive cognitive decline that is usually due to accumulating brain pathology. (p. 541)

Concept Check

1. Kelly, from earlier in this chapter, has an older brother, Kyle, who did not start speaking until he was nearly 20 months old. What factors might have contributed to such a difference between these children, assuming no diseases or disabilities?
2. A healthy 80-year-old businessman wants to continue working at his job, although he admits his memory is not quite what it used to be. What habits could he develop to help minimize his memory lapses?
3. Among tree lizards, some males are dominant and defend territories; others are nomadic foragers. Males that are given testosterone injections during the first 30 days of life are more likely to grow up to be dominant territory holders, but similar injections have no effect on adult non-dominant males. Why might this be?
4. “Antagonistic pleiotropy” is a formal name for the idea that a gene may be beneficial early in life (i.e., while an organism is still capable of reproducing) and yet have adverse consequences later in life. As long as the organism carrying the gene survives long enough to produce offspring, the gene can propagate through the population. Restate this general idea, using Alzheimer’s disease as an example. Can you think of specific examples from this chapter where processes that are beneficial early in life would be maladaptive if they continued into later life?

Answers to Test Your Knowledge

Learning and Memory in Old Age

1. This involves recalling an old episodic memory; recall of old, well-formed episodic memories tends *not* to decline much in healthy old age.
2. This involves working memory, which *does* tend to decline with old age.
3. This involves performance of a well-learned skill, which tends *not* to decline with healthy aging.
4. This involves acquiring new semantic information, an ability that *does* tend to decline with old age.
5. This involves learning a new skill, an ability that *does* tend to decline with old age.

Everything Grows Together ... at Different Rates

(Rough) Timetable for Development	Types of Learning and Memory	Critical Brain Area(s)
Present before birth (in at least some forms)	Sensory processing and habituation	Sensory cortex
Infancy to early childhood (in at least some forms)	Conditioning and skill learning	Cerebellum and basal ganglia
Childhood	Social learning and imitation	Mirror neurons in the motor cortex
Late childhood and early adolescence	Episodic memory and semantic memory	Hippocampus
Late adolescence and young adulthood	Working memory and executive function	Frontal cortex

GLOSSARY

acetylcholine (ACh)

A neuromodulator that strongly influences hippocampal function.

acoustic startle reflex

A defensive response (such as jumping or freezing) to a startling stimulus (such as a loud noise).

acquired equivalence

A learning and generalization paradigm in which prior training in stimulus equivalence increases the amount of generalization between two stimuli, even if those stimuli are superficially dissimilar.

activity-dependent enhancement

Paired training of CS and US that produces an increase in the glutamate vesicles released from sensory neurons to motor neurons.

adolescence

The transitional stage between puberty and adulthood, corresponding roughly to the teenage years in humans (ages 13 to 19).

allele

A naturally occurring variant of a gene.

altruism

In behavioral economics, an action or a behavior that provides benefit to another at the expense of some cost to the actor.

Alzheimer's disease

A form of dementia due to accumulating brain pathology (specifically, amyloid plaques and neurofibrillary tangles).

amnesia

Severe memory impairment.

amygdala

A collection of brain nuclei that lie at the anterior tip of each hippocampus; critical for learning and expressing emotional responses as well as mediating the emotional modulation of memory formation.

amyloid plaque

A deposit of beta-amyloid protein; these deposits accumulate in the brains of patients with Alzheimer's disease.

androgen

Any member of the principal class of sex hormones present in adult males.

anterograde amnesia

A severe loss of the ability to form new declarative memories.

apoptosis

Natural cell death, as opposed to cell death caused by accident or disease.

appetitive conditioning

Conditioning in which the US is a desirable event (such as food delivery).

arousal (fight-or-flight response)

A collection of bodily responses (including increased blood flow to muscles, increased respiration, and depressed digestion and immune function) that prepare the body to face a threat.

association cortex

Areas of the cerebral cortex involved in associating information within and across sensory modalities.

associationism

The principle that memory depends on the formation of linkages (“associations”) between pairs of events, sensations, and ideas, such that recalling or experiencing one member of the pair elicits a memory or anticipation of the other.

associative stage

The second stage in Fitts’s model of skill learning, when learners begin to use stereotyped actions in performing a skill and rely less on actively recalled memories of rules.

associative weight

In the Rescorla–Wagner model of conditioning, a value representing the strength of association between a conditioned stimulus (CS) and an unconditioned stimulus (US).

autism spectrum disorder

A developmental disorder associated with deficits in social interactions and social learning.

autonomic nervous system (ANS)

A collection of nerves and structures that control internal organs and glands.

autonomous stage

The third stage in Fitts’s model of skill learning, when a skill or subcomponents of the skill become motor programs.

aversive conditioning

Conditioning in which the US is a disagreeable event (such as a shock or an airpuff to the eye).

axon

The output extension of a neuron, specialized for transmitting signals to other neurons or to muscles.

basal forebrain

A collection of structures that lie at the base of the forebrain and are important in the production of acetylcholine that is distributed throughout the brain.

basal ganglia

A brain region that lies at the base of the forebrain and includes the dorsal striatum.

behavioral addiction

Pathological addiction to a behavior (rather than to a pharmacological substance).

behavioral economics

The study of how organisms allocate their time and resources among possible options.

behaviorism

A school of thought that says psychology should restrict itself to the study of observable behaviors (such as lever presses, salivation, and other measurable actions) and not seek to infer unobservable mental processes.

blind design

An experimental design in which the participants do not know the hypothesis being tested or whether they are, for example, receiving an experimental treatment or a placebo.

bliss point

In behavioral economics, the allocation of resources that maximizes subjective value or satisfaction.

blocking

A two-phase training paradigm in which prior conditioning with one cue (CS1 à US) blocks later learning of a second cue when the two are paired together in the second phase of the training (CS1 + CS2 à US).

brainstem

A group of structures that connects the rest of the brain to the spinal cord and plays key roles in regulating automatic functions such as breathing and body temperature.

Cannon–Bard theory of emotion

The theory that conscious feelings of emotion and bodily responses (physiological responses and overt behaviors) occur simultaneously and independently.

category

A division or class of entities in the world.

cell body

The central part of the prototypical neuron; it contains the nucleus and integrates signals from all the dendrites. Also known as the *soma*.

central executive

The component of Baddeley's model of working memory that monitors and manipulates the two working-memory buffers.

central nervous system (CNS)

The part of the vertebrate nervous system consisting of the brain and spinal cord.

cerebellum

A brain region that lies below the cerebral cortex in the back of the head. It is responsible for the regulation and coordination of complex voluntary muscular movement, including classical conditioning of motor-reflex responses.

cerebral cortex

The brain tissue covering the top and sides of the brain in most vertebrates; involved in storage and processing of sensory inputs and motor outputs.

chaining

An operant conditioning technique in which organisms are gradually trained to execute complicated sequences of discrete

responses.

chromosome

A packet of DNA; most human cells contain 23 pairs of chromosomes, with one member of each pair inherited from the mother and one from the father.

classical (Pavlovian) conditioning

A form of learning in which an animal acquires the expectation that a given stimulus predicts a specific upcoming important event.

closed skill

A skill that involves performing predefined movements that, ideally, never vary.

cochlear implant

A sensory prosthesis that directly stimulates auditory nerves to produce hearing sensations in deaf individuals.

cognitive approach

A subfield of psychology that emphasizes higher mental activities, such as language, memory, and perception.

cognitive control

The manipulation and application of working memory for planning, task switching, attention paying, stimulus selection, and the inhibition of inappropriate reflexive behaviors. Also known as *executive control* or *executive function*.

cognitive expectancy

The expected outcome of a response, used as a guide in determining whether or not to make that response.

cognitive map

An internal psychological representation of the spatial layout of the external world.

cognitive skill

A skill that requires problem solving or the application of strategies.

cognitive stage

The first stage in Fitts's model of skill learning, when an individual must actively think to encode and perform a skill.

compound conditioning

Conditioning in which two or more cues are present together, usually simultaneously, forming a compound CS.

concept formation

The process by which we learn about new categories of entities in the world, usually based on common features.

concept

An internal psychological representation of a real or abstract entity.

concurrent reinforcement schedule

A reinforcement schedule in which the organism can make any of several possible responses, each of which may lead to a different outcome (each of which may be reinforced according to a different reinforcement schedule).

conditioned avoidance

An experimental design in which animals learn to make particular responses to avoid or prevent exposure to an aversive stimulus.

conditioned emotional response

A classically conditioned response (CR), such as physiological arousal, that is produced in response to a CS that has been paired with an emotion-evoking US.

conditioned escape

An experimental design in which animals learn to make particular responses in order to escape from or terminate an aversive stimulus.

conditioned response (CR)

The trained response to a conditioned stimulus (CS) in anticipation of the unconditioned stimulus (US) that the CS predicts.

conditioned stimulus (CS)

A cue that is paired with an unconditioned stimulus (US) and comes to elicit a conditioned response (CR).

conditioned taste aversion

A conditioning preparation in which a subject learns to avoid a taste that has been paired with an aversive outcome, usually nausea.

confabulation

A behavior in which individuals, when asked to remember past events, respond with highly detailed but false memories; associated with some forms of *amnesia*.

confirmation bias

A tendency to ignore information that conflicts with a prior belief and focus on information that is consistent with that belief.

connectionist models

Models that represent learning and other thought processes as networks of connections between simple processing units called nodes.

consequential region

A set of stimuli in the world that share the same consequence as a stimulus whose consequence is already known.

consolidation period

A length of time during which new episodic and semantic memories are vulnerable and easily lost or altered; each time a memory is recalled, it may become vulnerable again until it has been “reconsolidated.”

constant practice

Practice involving a constrained set of materials and skills.

constraint-induced movement therapy

A motor-rehabilitation technique in which unaffected limbs are restrained to increase usage of dysfunctional limbs.

contiguity

Nearness in time (temporal contiguity) or space (spatial contiguity).

continuous reinforcement schedule

A reinforcement schedule in which every instance of the response is followed by the reinforcer.

copying

The act of doing what one observes another organism doing.

cortical plasticity

The capacity to change cortical organization as a result of experience.

cortisol

The chief glucocorticoid in humans.

CS modulation theory

Any of the theories of conditioning that say the stimulus that enters into an association is determined by a change in how the CS is processed.

cued recall

A memory test that involves some kind of prompt or cue to aid recall.

cumulative recorder

A device used for recording responses in operant conditioning, designed in such a way that the height of the line it draws represents the total (cumulative) number of responses made up to a given time.

data

Facts and figures from which conclusions can be inferred.

declarative memory

A broad class of memories, both semantic and episodic, that can typically be verbalized (“declared”) or explicitly communicated in some other way.

deep brain stimulation

A procedure that involves delivering an electrical current into a patient’s brain through one or more implanted electrodes; used to alleviate tremors and other motor symptoms associated with Parkinson’s disease.

delay conditioning

A conditioning procedure in which there is no temporal gap between the end of the CS and the beginning of the US and in which the CS co-terminates with the US.

delay discounting

The progressive reduction (or discounting) of the subjective value of a reward the longer it is delayed.

delayed nonmatch-to-sample (DNMS) task

A test of visual memory in which a subject must indicate which of two objects is not the same as the sample object that was recently seen.

dementia

Progressive cognitive decline, usually due to accumulating brain pathology.

dendrite

On a neuron, an extension that is specialized to receive signals from other neurons.

dependent variable

In an experiment, the factor whose change is measured as an effect of changes in the independent variable.

depression

A psychiatric condition that involves sadness as well as a general loss of initiative and activity.

differential reinforcement of alternative behaviors (DRA)

A method used to decrease the frequency of unwanted behaviors by instead reinforcing preferred alternative behaviors.

diffusion tensor imaging (DTI)

A type of MRI that measures connections between brain regions.

directed forgetting

A procedure in which participants are first asked to learn information and later asked to remember or forget specific items; typically, memory is worse for items that a participant was directed to forget.

direct-matching hypothesis

The proposal that memories for actions are stored in specialized cortical regions that map observed actions onto the motor representations of the actions.

discrete-component representation

A representation in which each individual stimulus (or stimulus feature) corresponds to one element (node) in the model.

discrete-trials paradigm

An operant conditioning paradigm in which the experimenter defines the beginning and end points of each trial.

discrimination learning

The process by which animals or people learn to respond differently to different stimuli.

discriminative stimulus (S^D)

In operant conditioning, a stimulus indicating that a particular response (R) may lead to a particular outcome (O).

dishabituation

A renewal of a response, previously habituated, that occurs when the organism is presented with a novel stimulus.

distributed representation

A representation in which information is coded as a pattern of activation distributed across many different nodes.

DNA

(formally, *deoxyribonucleic acid*) The genetic material found within all living cells that contains the instructions for making an organism.

dopamine

A neuromodulator that alters neuron-to-neuron communication.

dorsal anterior cingulate cortex (dACC)

A brain region that may play a role in the motivational value of pain.

dorsal striatum

A region of the basal ganglia that is important for stimulus–response learning.

dorsolateral prefrontal cortex (DLPFC)

The left and right sides of the topmost part of the prefrontal cortex (PFC).

double-blind design

An experimental design in which neither the experimenters nor the subjects know group assignment.

Down syndrome

A congenital disorder that causes mild-to-moderate intellectual disability and is usually due to trisomy 21.

drive reduction theory

The theory, proposed by Clark Hull, that all learning reflects the innate, biological need to obtain primary reinforcers.

drug

A chemical substance that alters the biochemical functioning of the body and, in many cases, affects the brain.

dual process theory

The theory that habituation and sensitization are independent of each other but operate in parallel.

dualism

The principle that the mind and body exist as separate entities.

dysexecutive syndrome

A disrupted ability to think and plan.

echolalia

The automatic repetition of words or phrases immediately after hearing them spoken.

electroconvulsive shock

A brief pulse of electricity that is passed through the brain and can severely disrupt newly formed memories.

electrodermal activity (EDA)

Fluctuations in the electrical properties of a person's skin that are a consequence of activity in the peripheral nervous system.

electroencephalography (EEG)

A method for measuring electrical activity in the brain by means of electrodes placed on the scalp; the resulting image is an *electroencephalogram* (also *EEG*).

elicited imitation

A technique for assessing memory in infants by observing their ability to mimic actions they have seen earlier.

emotion

A cluster of three distinct but interrelated sets of phenomena—physiological responses, overt behaviors, and conscious feelings—

produced in response to an affecting situation.

emotional contagion

An inborn tendency to react emotionally to visual or acoustic stimuli that indicate an emotional response by other members of one's species, typically in ways that replicate the observed response.

empiricism

A philosophical school of thought that holds that all the ideas we have are the result of experience.

emulation

Copying that involves replicating an outcome without replicating specific motor acts.

encoding specificity effect

The principle that retrieval is likely to be more successful if the cues and contextual conditions present at recall are similar to those that were present at encoding.

endogenous opioid

Any of a group of naturally occurring neurotransmitter-like substances that have many of the same effects as heroine, morphine, and other opiate drugs; may help signal the hedonic value of reinforcers in the brain.

engram

A physical change in the brain that forms the basis of a memory.

enriched environment

An environment that provides sensory stimulation and opportunities to explore and learn; for a rat, this may mean housing in a large cage with many toys to play with and other rats to socialize with.

epigenetics

The study of mechanisms by which gene expression can be modified (without modifying the genes themselves).

epinephrine (adrenaline)

A stress hormone that helps mediate the fight-or-flight response.

episodic memory

Memory for personal experience of specific autobiographical events; it includes information about the spatial and temporal contexts in which the event occurred.

error-correction learning

Learning through trial and error to reduce the discrepancy (error) between what is predicted and what actually occurs.

errorless discrimination learning

A training procedure in which a difficult discrimination is learned by starting with an easy version of the task and proceeding to incrementally harder versions as the easier ones are mastered.

estrogen

Any member of the principal class of sex hormones present in adult females.

event-related potential (ERP)

Electroencephalograms (EEGs) from a single individual averaged over multiple repetitions of an event (such as a repeated stimulus presentation).

evolution

The change that species undergo over time, with new traits or characteristics being passed from one generation to the next; natural selection is one mechanism by which evolution occurs.

experimenter bias

The influence an experimenter's prior knowledge or expectations can have (consciously or unconsciously) on the outcome of an experiment.

expert

A person who performs a skill better than most.

explicit memory

A category of memory that includes semantic memory and episodic memory and that consists of memories of which the person is aware: you know that you know the information.

extinction

The process of reducing a learned response to a stimulus by ceasing to pair that stimulus with a reward or punishment.

eyeblink conditioning

A classical conditioning procedure in which the US is an airpuff to the eye and the conditioned and unconditioned responses are eyeblinks.

false memory

Memory for events that never actually happened.

familiarity

The perception of similarity that occurs when an event is repeated.

fear response

A cluster of physiological changes, overt behaviors, and conscious reactions that accompany the emotion of fear. In the laboratory, the physiological changes and motor behaviors are often taken to imply presence of fear whether or not the accompanying conscious experience of fear can be documented.

fixed-interval (FI) schedule

A reinforcement schedule in which the first response after a fixed amount of time is reinforced; thus, FI 1-min means that reinforcement follows the first response made after a 1-minute interval since the last reinforcement.

fixed-ratio (FR) schedule

A reinforcement schedule in which a specific number of responses must occur before a reinforcer is delivered; thus, FR 5 means that reinforcement arrives after every fifth response.

flashbulb memory

A memory formed under conditions of extreme emotions that seems especially vivid and long-lasting.

forgetting

The loss or deterioration of memory over time.

fornix

A fiber bundle that connects subcortical structures, including the basal forebrain, to the hippocampus.

free recall

A memory test that involves simply generating requested information from memory.

free-operant paradigm

An operant conditioning paradigm in which the animal can operate the apparatus as it chooses in order to obtain reinforcement (or avoid punishment).

frontal cortex

The regions of cortex that lie within the frontal lobes and that may play a role in determining which memories are stored and in producing metamemory for that information.

frontal lobe

The part of the cerebral cortex lying at the front of the human brain; enables a person to plan and perform actions.

functional amnesia

A sudden retrograde memory loss that seems to result from psychological causes rather than physical causes such as brain injury; also called *psychogenic amnesia*.

functional magnetic resonance imaging (fMRI)

A method of functional neuroimaging based on comparing an MRI of the brain during performance of a task with an MRI of the brain at rest.

functional neuroimaging

Techniques (such as fMRI) for observing the activity or function of a living brain.

gene

A segment of a DNA strand that contains information for building a protein from amino acids; in essence, genes are the recipes for constructing and maintaining an organism.

generalization

The transfer of past learning to novel events and problems.

generalization gradient

A curve showing how physical changes in stimuli (plotted on the horizontal axis) correspond to changes in behavioral responses (plotted on the vertical axis).

gestational age

Time since conception.

glia

Cells of various types that provide functional or structural support to neurons; some contribute to changes in connections between neurons.

glucocorticoid

Any of a class of stress hormones (including cortisol in humans) that help to mediate the fight-or-flight response.

habituation

A decrease in the strength or occurrence of a behavior after repeated exposure to the stimulus that produces that behavior.

Hebbian learning

The principle that learning involves strengthening the connections of coactive neurons; often stated as, “Neurons that fire together, wire together.”

hedonic value

In operant conditioning, the subjective “goodness” or value of a reinforcer.

heterosynaptic

Occurring in several nearby synapses simultaneously.

hippocampal region

The hippocampus and associated brain regions, including the entorhinal cortex and dentate gyrus. In humans, also referred to as the *medial temporal lobe*.

hippocampus

A brain structure located in the medial temporal lobe that is important in the formation of new memories.

homeostasis

The tendency of the body (including the brain) to gravitate toward a state of equilibrium or balance.

homosynaptic

Occurring in one synapse without affecting nearby synapses.

hypothesis

A supposition made on the basis of limited evidence as a starting point for further investigation.

identical elements theory

Thorndike’s proposal that learned abilities transfer to novel situations to an extent that depends on the number of elements in the new situation that are identical to those in the situation in which the skills were encoded.

implicit learning

Learning that occurs without the learner’s awareness of improvements in performance or, in the case of people with amnesia, awareness that practice has occurred.

implicit memory

Memory that occurs without the learner’s awareness.

imprinting

The tendency of young animals of some species to form an attachment to the first individual they see after birth.

incentive salience hypothesis

The hypothesis that dopamine helps provide organisms with the motivation to work for reinforcement.

independent variable

The factor that is manipulated in an experiment.

inductive inference

A logical inference that is probably (but not necessarily) true and is usually based on attempts to draw a general rule from one or more specific instances or premises.

inferior olive

Located in the brainstem, a nucleus of cells that conveys information about aversive stimuli, such as an airpuff US, up to both the interpositus nucleus and the cerebellar cortex.

insular cortex (insula)

A brain region that is involved in conscious awareness of bodily and emotional states and that may play a role in signaling the aversive value of stimuli.

interference

Reduction in the strength of a memory due to overlap with the content of other memories.

interpositus nucleus

One of the cerebellar deep nuclei from which the conditioned response output is generated in classically conditioned motor responses.

interstimulus interval (ISI)

The temporal gap between the onset of the CS and the onset of the US.

James–Lange theory of emotion

The theory that conscious feelings of emotion occur when the mind senses the physiological responses associated with fear or some other kind of arousal.

knowledge of results

Feedback about performance of a skill, critical to the effectiveness of practice.

latent inhibition

A conditioning paradigm in which prior exposure to a CS retards later learning of the CS–US association during acquisition training.

latent learning

Learning that is unconnected to a positive or negative consequence and that remains undetected (latent) until explicitly demonstrated at a later stage.

law of effect

The observation, made by Thorndike, that the probability of a particular behavioral response increases or decreases depending on the consequences that have followed that response.

learned helplessness

A phenomenon in which exposure to an uncontrollable punisher teaches an expectation that responses are ineffectual, which in turn reduces the motivation to attempt new avoidance responses.

learning

The process by which changes in behavior arise as a result of experiences interacting with the world.

learning curve

A graph showing learning performance (the dependent variable, usually plotted along the vertical axis) as a function of training time (the independent variable, usually plotted along the horizontal axis).

learning set formation

Acquisition of the ability to learn novel tasks rapidly based on frequent experiences with similar tasks.

lesion

Damage caused by injury or illness.

levels-of-processing effect

The finding that, in general, deeper processing (such as thinking about the semantic meaning of a word) leads to better recall of the information than shallow processing (such as thinking about the spelling or pronunciation of a word).

long-term depression (LTD)

A process in which synaptic transmission becomes less effective as a result of recent activity; with long-term potentiation, LTD is widely believed to represent a form of synaptic plasticity that could be the neural mechanism for learning.

long-term memory (LTM)

Permanent or near-permanent storage of memory that lasts beyond a period of conscious attention.

long-term potentiation (LTP)

A process in which synaptic transmission becomes more effective as a result of recent activity; with long-term depression, LTP is widely believed to represent a form of synaptic plasticity that could be the neural mechanism for learning.

magnetic resonance imaging (MRI)

A method of structural neuroimaging based on recording changes in magnetic fields.

massed practice

Concentrated, continuous practice of a skill.

matching law of choice behavior

The principle that an organism, given a choice between multiple responses, will make each response at a rate proportional to how often that response is reinforced relative to the other choices.

mathematical psychology

A subfield of psychology that uses mathematical equations to describe the laws of learning and memory.

medial temporal lobes

The medial (or inner) surface of the temporal lobes that contains the hippocampus, the amygdala, and other structures important for memory.

memory

The internal record of past experiences acquired through learning.

metamemory

Knowledge of, and ability to think about, our own memories, including both feeling of knowing and judgment of learning.

mirror neuron

A neuron that responds during performance of an action and also during observations of that action.

mirror reading

An experimental task that requires individuals to read mirror-reversed text; used to test cognitive skill learning.

mirror tracing

An experimental task that requires individuals to trace drawings by watching a mirror image of their hand and of the figure to be traced, with the actual hand and figure concealed; used to test perceptual-motor skill learning.

modeling

Demonstration of actions.

mood congruency of memory

The principle that it is easiest to retrieve memories that match our current mood or emotional state.

motivational value

In operant conditioning, the degree to which an organism is willing to work to obtain access to a stimulus.

motor program

A sequence of movements that an organism can perform automatically (with minimal attention).

motor prosthesis

An electromechanical device that can help a person recover lost abilities to learn and perform perceptual-motor skills.

multiple trace theory

The theory that episodic (and possibly semantic) memories are encoded by an ensemble of hippocampal and cortical neurons and that both the hippocampus and cortex are normally involved in storing and retrieving even very old memories.

nativism

A philosophical school of thought that holds that the bulk of knowledge is inborn (or native).

natural selection

A proposed mechanism for evolution, also known as “survival of the fittest,” which holds that species evolve when there is some trait that is inheritable, varies naturally across individuals, and increases an individual’s “fitness,” or chance of survival and reproductive success.

negative contrast

The phenomenon in which the reinforcing value of one reward is reduced because a better reward is expected.

negative patterning

A behavioral paradigm in which the appropriate response to individual cues is positive, whereas the appropriate response to their combination (pattern) is negative (no response).

negative punishment

A type of operant conditioning in which the response causes a desirable element to be “subtracted from” the environment; over time, the response becomes less frequent.

negative reinforcement

A type of operant conditioning in which the response causes an undesirable element to be “subtracted from” the environment; over time, the response becomes more frequent.

nervous system

An organism’s system of tissues specialized for monitoring sensations, generating movements, and maintaining the function of most internal organs.

neural connectivity

The connections between neurons through which brain systems interact.

neurofibrillary tangle

An aggregation of collapsed remains of the tau proteins that normally hold a neuron in place and help transport nutrients around the cell; such tangles occur in the brains of patients with Alzheimer’s disease.

neurogenesis

Creation of new neurons in the brain.

neuromodulator

A neurotransmitter that acts to modulate activity in a large number of neurons rather than in a single synapse.

neuron

A special type of cell that is one of the main components of the nervous system.

neurophysiology

The study of the activity and function of neurons.

neuropsychology

The branch of psychology that deals with the relationship between brain function and behavior.

neuroscience

The study of the brain and the rest of the nervous system.

neurotransmitter

One of several classes of molecule released by neurons to carry chemical messages to other neurons.

nondeclarative memory

A broad class of memory that includes skill memory and other types of learning that do not fall under either the episodic or semantic memory categories and that are not always consciously accessible or easy to verbalize.

non-REM sleep

A category of sleep that includes both light sleep and slow-wave sleep (SWS).

norepinephrine

A neurotransmitter that is chemically related to epinephrine but that (unlike epinephrine) can cross the blood–brain barrier and

affect brain function.

novel object recognition task

A task in which an organism's detection of and response to unfamiliar objects during exploratory behavior are used to measure its memories of past experiences with those objects.

nucleus basalis

A small group of neurons located in the basal forebrain; these neurons deliver acetylcholine to the cortex, enabling cortical plasticity.

observational conditioning

A process in which an individual learns an emotional response after observing similar responses in others.

occipital lobe

The part of the cerebral cortex lying at the rear of the human brain; important for visual processing.

open skill

A skill in which movements are made on the basis of predictions about changing demands of the environment.

operant conditioning

The process whereby organisms learn to make or to refrain from making certain responses in order to obtain or avoid certain outcomes.

optogenetics

A technique for causing specific neurons or other cells to become sensitive to light, after which researchers can use light stimulation to turn those cells "on" and "off" at will.

orbitofrontal cortex

A brain region that is important for learning to predict the outcomes of particular responses.

orienting response

An organism's innate reaction to a novel stimulus.

overshadowing

An effect seen in compound conditioning when a more salient cue within a compound acquires more association strength than does the less salient cue and is thus more strongly associated with the US.

parietal lobe

The part of the cerebral cortex lying at the top of the human brain; important for processing somatosensory (touch) inputs.

Parkinson's disease

A disorder resulting from disruptions in the normal functioning of the basal ganglia and involving progressive deterioration of motor control and perceptual-motor skill learning.

partial reinforcement schedule

A reinforcement schedule in which only some instances of the response are followed by the reinforcer.

pathological addiction

A strong habit that is maintained despite harmful consequences.

perceptual learning

Learning in which experience with a set of stimuli makes it easier to distinguish those stimuli.

perceptual-motor skill

Learned movement patterns guided by sensory inputs.

peripheral nervous system (PNS)

The part of the nervous system that transmits signals from sensory receptors to the central nervous system and carries commands from the CNS to muscles.

perseveration

A failure to learn a new response, especially as demonstrated by continued adherence to an old response rule that is no longer valid.

perspective taking

Imagining oneself in the place of another.

phobia

An excessive and irrational fear of an object, a place, or a situation.

phonological loop

The component of Baddeley's model of working memory that maintains auditory memories by internal (subvocal) speech rehearsal.

phrenology

An obsolete field of study that attempted to determine mental abilities by measuring head shape and size.

piloerection

A fear response in mammals in which body hair stands on end, making the animal look bigger and more threatening than it is.

place cell

A neuron that fires maximally when the organism enters a particular location within an environment.

placebo

An inactive substance, such as a sugar pill, that is administered to one group of participants in an experiment to compare against the effects of an active substance, such as a drug.

plasticity

The capacity of brain structure to change over time.

positive punishment

A type of operant conditioning in which the response causes an undesirable element to be "added" to the environment; over time, the response becomes less frequent.

positive reinforcement

A type of operant conditioning in which the response causes a desirable element to be "added" to the environment; over time, the

response becomes more frequent.

post-reinforcement pause

In operant conditioning, a brief pause in responding that follows delivery of the reinforcer.

postsynaptic

On the receiving side of a synapse.

posttraumatic stress disorder (PTSD)

A psychological syndrome that can develop after exposure to a traumatic event (e.g., combat, rape, natural disaster); symptoms include re-experiencing the event (through intrusive recollections, flashbacks, or nightmares), avoidance of reminders of the trauma, emotional numbing, and heightened anxiety.

power law of practice

A law stating that the degree to which each new practice session improves performance diminishes after a certain point, such that greater numbers of sessions are needed to further improve the skill; learning occurs quickly at first and then slows down.

prediction error

The difference between what was predicted and what actually occurred.

prefrontal cortex (PFC)

The frontmost (anterior) part of the frontal-lobe cortex, essential for working memory and executive control.

presynaptic

On the sending side of a synapse.

primary reinforcer

A reinforcer, such as food, water, or sleep, that is of biological value to an organism.

priming

A phenomenon in which prior exposure to a stimulus can improve the ability to recognize that stimulus later.

proactive interference

Disruption of new learning by previously stored information.

prototype

The central tendency or idealized version of a category.

psychology

The study of mind and behavior.

puberty

The process of physical change during which the body transitions to sexual maturity, usually beginning around age 10 in human females and age 12 in human males.

punisher

A consequence of behavior that leads to decreased likelihood of that behavior in the future.

punishment

The process of providing outcomes (punishers) that lead to decreased probability of a particular behavior occurring in the future.

Purkinje cell

A type of large, drop-shaped, and densely branching neuron in the cerebellar cortex.

radical behaviorism

An extreme form of behaviorism championed by B. F. Skinner and holding that consciousness and free will are illusions and that even so-called higher cognitive functions (e.g., human language) are merely complex sets of stimulus–response associations.

rapid eye movement (REM) sleep

A phase of sleep in which the eyes move rapidly under closed lids.

receptive field

The range (or “field”) of physical stimuli that activate a single neuron.

receptor

A specialized molecule, located on the surface of a neuron, to which one or more particular neurotransmitters can bind; when a neurotransmitter activates a receptor, effects may be initiated in the neuron.

reciprocal altruism

In behavioral economics, the principle that one organism may donate time or resources to help another in the expectation that the other will return the favor later on.

recognition

A memory test that involves picking out (or recognizing) a studied item from a set of options.

reconsolidation

The process whereby each time an old memory is recalled or reactivated, it may become vulnerable to modification.

reflex

An involuntary and automatic (unlearned) response.

reflex arc

An automatic pathway from a sensory stimulus to a motor response.

reinforcement schedule

A schedule that determines how often reinforcement is delivered in an operant conditioning paradigm.

reinforcement

The process of providing outcomes (reinforcers) that lead to increased probability of a particular behavior occurring in the future.

reinforcer

A consequence of behavior that leads to increased likelihood of that behavior in the future.

response

The behavioral consequence of perception of a stimulus.

retention curve

A graph showing forgetting or relearning as a function of time since initial learning.

retroactive interference

Disruption of old (previously stored) information by more recent learning.

retrograde amnesia

Loss of memories for events dating from before a brain injury or disruption; the memory loss is generally time graded, with more recent memories devastated and older ones being spared.

Ribot gradient

A pattern of retrograde memory loss in which recently acquired memories are more prone to disruption than older memories.

rotary pursuit task

An experimental task that requires individuals to keep the end of a pointed stick (stylus) above a fixed point on a rotating disk; used to study perceptual-motor skill learning.

secondary reinforcer

A reinforcer that initially has no biological value but that has been paired with (or predicts the arrival of) a primary reinforcer.

self-control

An organism's willingness to forgo a small immediate reward in favor of a larger future reward.

semantic memory

Memory for facts or general knowledge about the world, including general personal information.

sensitive period

A time window, usually early in life, during which a certain kind of learning occurs most easily and is most effective.

sensitization

A phenomenon in which a salient stimulus (such as an electric shock) temporarily increases the strength of responses to other stimuli.

sensory cortex

Areas of the cerebral cortex involved in processing sensory information such as sight and sound.

sensory memory

Brief, transient sensations of what has just been perceived when someone sees, hears, or tastes something.

sensory preconditioning

Training in which presentation of two stimuli together as a compound results in a later tendency to generalize what is known about one of these stimuli to the other.

sensory prosthesis

A mechanical device designed to supplement or substitute for a faulty sensory modality such as vision or hearing; the device's sensory detectors interface with brain areas that normally process those sensory inputs.

serial reaction time task

An experimental task that requires individuals to press keys in specific sequences on the basis of cues provided by a computer; used to study implicit learning.

shaping

An operant conditioning technique in which successive approximations to the desired response are reinforced.

short-term memory (STM)

A temporary memory that is maintained through active rehearsal.

single-cell recording

Use of an implanted electrode to detect electrical activity (spiking) in a single cell (such as a neuron).

skill

An ability that can improve over time through practice.

skill decay

Loss of a skill due to non-use.

Skinner box

A chamber used for operant conditioning and designed so that reinforcement or punishment is delivered automatically whenever an animal makes a particular response (such as pressing or releasing a lever).

slow-wave sleep (SWS)

A phase of sleep characterized by large, slow oscillations in the brain that are highly synchronized over wide brain areas.

social conformity

The tendency to adopt the behavior of the group.

social learning

Learning from others through observation or communication.

social learning theory

A theory of human behavior, prominent from the 1940s through the 1960s, proposing that the kinds of reinforcements an individual has experienced in past social contexts will determine how that individual will act in any given situation.

social transmission of information

A process in which an individual directly or indirectly communicates information that may affect the future actions of group members.

somatic theories of emotion

Theories of emotion based on the central premise that physiological responses to stimuli come first, and these determine or induce emotions.

source monitoring error

Remembering information but being mistaken about the specific episode that is the source of that memory.

spaced practice

Practice of a skill that is spread out over several sessions.

spatial learning

The acquisition of information about one's surroundings.

spine

A tiny protrusion on a dendrite where synapses may form.

spontaneous recovery

Reappearance (or increase in strength) of a previously habituated response after a short period of no stimulus presentation.

standard consolidation theory

The theory that the hippocampus and related medial temporal lobe structures are required for storage and retrieval of recent episodic and semantic memories but not older ones.

statistical learning

Learning driven by repeated exposures to perceptual events that increases the familiarity and distinctiveness of those events.

stereotype

A set of beliefs about the attributes of the members of a group.

stimulus

A sensory event that provides information about the outside world.

stimulus control

The influence of cues in the world on an organism's behavior.

stimulus enhancement

A process in which observations of other individuals draw an organism's attention toward specific objects, events, or locations within an environment.

stimulus representation

The form in which information about stimuli is encoded within a model or brain.

stress

Any stimulus or event that causes bodily arousal and release of stress hormones.

stress hormone

A hormone that is released in response to signals from the autonomic nervous system (ANS) and helps mediate the fight-or-flight response; examples include norepinephrine and the glucocorticoids.

stroke

An event in which blood flow to some region of the brain stops or in which an artery ruptures, causing neurons in the affected region to die.

structural neuroimaging

Techniques (such as MRI) for creating images of anatomical structures within the living brain.

subject bias

The influence a subject's prior knowledge or expectations can have (consciously or unconsciously) on the outcome of an

experiment.

substantia nigra pars compacta (SNc)

A part of the basal ganglia that contains dopamine-producing neurons projecting to the striatum.

synapse

A narrow gap between two neurons, across which chemical messages can be transmitted.

synaptic depression

A reduction in synaptic transmission; a possible neural mechanism underlying habituation.

synaptic plasticity

The ability of synapses to change as a result of experience.

synaptogenesis

Creation of new synapses.

systematic desensitization

Therapy for phobias in which successive approximations of the fear-evoking stimulus are presented while the patient learns to remain relaxed; eventually, even presentation of the stimulus itself does not elicit a fear reaction.

talent

A person's genetically endowed ability to perform a skill better than most.

template model of song learning

A model of song learning that has three basic phases: song memorization, song practice, and song utilization.

temporal lobe

The part of the cerebral cortex lying at the sides of the human brain; important for language and auditory processing and for learning new facts and forming new memories of events.

tend-and-befriend response

A response to threat that involves tending to offspring (tending) and seeking out social support (befriending).

testosterone

The most important androgen and hence the principal male sex hormone.

theory

A set of statements devised to explain a group of facts.

theory of equipotentiality

The theory that memories are stored globally by the brain as a whole rather than in one particular brain area.

token economy

An environment (such as a prison or schoolroom) in which tokens function the same way as money does in the outside world.

tolerance

A decrease in reaction to a drug such that larger doses are required to achieve the same effect.

trace conditioning

A conditioning procedure in which there is a temporal gap between the end of the CS and the beginning of the US.

transfer of training

The transfer of skill memories to novel situations.

transfer specificity

The restricted applicability of learned skills to specific situations.

transfer-appropriate processing (TAP) effect

The principle that memory retrieval will be best when the way that information is processed at encoding matches how it will be processed at retrieval.

transient global amnesia (TGA)

A transient, or temporary, disruption of memory typically including elements of both anterograde and retrograde amnesia.

transient memory

Nonpermanent memory that lasts seconds or minutes; the Atkinson–Shiffrin model describes two types: sensory and short-term memory.

trial-level model

A theory of learning in which all the cues that occur during a trial and all the changes that result are considered a single event.

trisomy 21

A condition in which an embryo inherits three (rather than two) copies of chromosome 21, resulting in Down syndrome.

true imitation

Copying that involves reproducing motor acts.

two-action test

A technique developed to investigate imitation abilities; it involves exposing naive animals to demonstrators trained to achieve the same goal using different actions.

two-factor theory of avoidance learning

A theory that avoidance involves an interaction between classical conditioning and operant conditioning.

two-factor theory of emotion

The theory that a combination of cognitive appraisal and perception of biological changes together determines our conscious feelings of emotion.

unconditioned response (UR)

The naturally occurring response to an unconditioned stimulus (US).

unconditioned stimulus (US)

A cue that has some biological significance and that, in the absence of prior training, naturally evokes a response.

US modulation theory

Any of the theories of conditioning that say the stimulus that enters into an association is determined by a change in how the US

is processed.

variable practice

Practice involving the performance of skills in a wide variety of contexts.

variable-interval (VI) schedule

A reinforcement schedule in which the first response after a fixed amount of time, on average, is reinforced; thus, VI 1-min means that reinforcement follows the first response made after a 1-minute interval, on average, since the last reinforcement.

variable-ratio (VR) schedule

A reinforcement schedule in which a specific number of responses, on average, must occur before a reinforcer is delivered; thus, VR 5 means that, on average, every fifth response is reinforced.

ventral tegmental area (VTA)

A brain region that contains dopamine-producing neurons projecting to the frontal cortex and other brain areas.

ventrolateral prefrontal cortex (VLPFC)

The lower left and right sides of the PFC.

visual sensory memory

The initial temporary storage for information perceived by the visual system.

visuospatial sketchpad

The component of Baddeley's model of working memory that holds visual and spatial images for manipulation.

vocal imitation

Copying that involves vocal reproduction of sounds.

vocal learning

Modifying one's vocal outputs on the basis of sounds one has heard.

word-length effect

The tendency for a person to remember fewer words from a list as the length of the words increases.

word-stem completion task

A task in which participants are asked to fill in the blanks in a list of word stems (e.g., MOT__) to produce the first word that comes to mind; in a priming experiment, participants are more likely to produce a particular word (e.g., MOTEL) if they have been exposed to that word previously.

working memory

The active and temporary representation of information that is maintained for the short term, available for manipulation.

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NAME INDEX

A

Aaron, L., [545](#)
Abbott, P., [320](#)
Abdel-Azim, E., [210](#)
Abdolmohommadi, B., [319](#)
Abe, N., [322](#)
Abel, T., [109](#)
Aberman, J., [210](#), [211](#)
Abraham, C., [535](#)
Abrams, T. W., [77](#), [167](#)
Acker, J. D., [533](#)
Ackerman, P. L., [330](#), [340](#), [343](#)
Ackermann, H., [158](#)
Acosta, A., [422](#)
Acredolo, L. P., [512](#)
Adamkiewicz, E., [184](#)
Adams, C., [210](#)
Adams, M. J., [285](#)
Adams, M. M., [357–358](#)
Adams, R. B., Jr., [422](#)
Ader, R. J., [168](#)
Adishesan, A., [203](#)
Adishesan, T., [203](#)
Adolph, K. E., [345](#)
Adolphs, R., [442](#), [445](#)
Aerni, A., [521](#)
Aharon, I., [219](#)
Ahern, F., [523](#), [526](#)
Ahmadinejad, M., [295](#)
Aidman, E. V., [460](#)
Aisen, P. S., [544](#)
Akeson, E., [540](#)
Akey, L., [210](#)

Akins, C. K., [473](#)
Alberici, E., [321](#)
Alberini, C. M., [42](#), [70](#)
Albohn, D. N., [422](#)
Alborn, A., [537](#)
Alexander, G. E., [395](#)
Alexander the Great, [1](#)
Alford, K. A., [539](#)
Alivisatos, B., [400](#)
Alkire, M., [435](#), [447](#), [450](#)
Allard, E. S., [422](#)
Allen, M. T., [147](#), [159](#), [161](#), [265](#), [266](#)
Allison, J., [199](#)
al Maskati, H., [447](#)
Alpern, G. D., [494](#)
Alsop, D. C., [408](#)
Alter, K., [428](#)
Alvarado, M. D., [109](#)
Alvarez, P., [313](#)
Alvarez, V. E., [319](#)
Alvarez-Buylla, A., [537](#)
Alvarez-Pelaez, R., [441](#)
Alves, S., [460](#)
Alzheimer, A., [541](#)
Alzheimer's Association, [544](#), [545](#)
Alzheimer's Disease Neuroimaging Initiative (ADNI), [542](#)
Amaral, D. G., [277](#), [305](#)
Amato, L., [220](#)
Amen, D. G., [393](#)
Amsel, R., [393](#)
Amso, D., [506](#)
An, D. H., [202](#)
An, Y. R., [202](#)
Anasari, A., [189](#)
Anderson, C. A., [252](#), [486](#), [487](#)
Anderson, I. M., [201](#), [207](#)

Anderson, J. R., [280](#), [293](#), [336](#)
Anderson, M. C., [292](#), [306](#), [457](#), [458](#), [518](#)
Anderson, P., [488](#)
Andersson, F., [489](#)
Andrykowski, M. A., [169](#)
Angus, K., [488](#)
Aniston, J., [302](#)
Annese, J., [305](#)
Apostolova, L. G., [543](#)
Appel, J., [543](#)
Aquirre, G. K., [405](#)
Araque, A., [42](#)
Araujo Filho, G. M., [456](#)
Araujo Nego, C. A., [456](#)
Arbisi, P. A., [461](#)
Archibald, S. J., [493](#)
Argolo, F. C., [458](#)
Aristophanes, [222](#)
Aristotle, [1–3](#), [10](#), [135](#), [331](#)
Arizzi, M., [210](#), [211](#)
Arnold, S., [546](#)
Arnsten, A. F., [411](#)
Aron, A. P., [423](#)
Aronson, J., [252](#)
Arthur, W., [336](#), [345](#)
Ashburner, J., [44](#)
Ashby, F. G., [205](#)
Ashmore, R. D., [252](#)
Asken, B. M., [319](#)
Astur, R., [514](#), [531](#)
Atalay, M., [545](#)
Atkinson, R., [370](#)
Atlas, S., [408](#)
Avikainen, S., [495](#)
Awh, E., [379](#)
Axelrod, R., [203](#)

Azpurua, J., [536](#)

B

Baamonde, C., [540](#)
Babinsky, R., [434](#), [435](#)
Bach, H., [175](#), [186](#)
Bach, J. S., [22](#), [515](#)
Bacher, D., [364](#)
Bachevalier, J., [109](#), [517](#), [530](#)
Baddeley, A. D., [285](#), [288](#), [336](#), [374–375](#), [379–380](#), [381](#), [382](#), [387](#), [390](#), [399](#), [400](#), [401](#), [518](#)
Badre, D., [402](#)
Bahrack, H., [289](#)
Bahrack, P., [289](#)
Bailey, C. H., [165](#), [542](#)
Bailey, J. S., [190](#)
Bailey, P., [376](#)
Bak, R., [455](#)
Baker, A. J., [483](#)
Bakin, J. S., [262](#), [263](#)
Baldo, J. V., [393](#)
Baldwin, T. T., [346](#)
Balkan, B., [515](#)
Ball, G., [536](#)
Balleine, B. W., [206](#), [432](#), [442](#)
Bancaud, J., [441](#)
Bandettini, P., [303](#)
Bandura, A., [469–471](#), [474](#), [496](#)
Banerjee, P., [546](#)
Bao, J. X., [164](#)
Bao, S., [158](#)
Baptista, L. F., [482](#)
Bar, S. K., [299](#)
Barale, F., [494](#)
Barbeau, E. J., [110](#)
Barch, D. M., [407](#), [408](#)

Barclay, T., [546](#)
Bard, K. A., [472–473](#), [474](#)
Bard, P., [423](#)
Barnes, C. A., [109](#), [535](#), [536](#)
Barnes, L. L., [515](#)
Barnes, T. D., [349](#)
Barnfield, A., [514](#)
Baron-Cohen, S., [494](#)
Barone, P., [219](#)
Barr, R., [516](#)
Barry, R. J., [77](#)
Barthelemy, C., [489](#)
Bartolomei, F., [110](#)
Barton, S., [494](#)
Bartsch, D., [165](#)
Basak, C., [351](#)
Basham, M. E., [492](#)
Bass, K., [525](#)
Bastien, C., [78](#)
Battaglia, G., [349](#)
Bauer, P., [506](#)
Bauer, R. M., [319](#)
Baum, W. M., [201](#)
Baumann, S., [428](#)
Bavaresco, J., [288](#)
Bavelier, D., [352](#)
Baxter, D., [210](#)
Baxter, M. G., [533](#)
Bayer, A., [542](#)
Beadle-Brown, J. D., [494](#)
Bear, M. F., [110](#)
Beaunieux, H., [361](#)
Bechara, A., [214](#), [218](#), [442](#), [445](#)
Beck, B., [333](#)
Beck, H. P., [454](#)
Beck, K. D., [532](#)

Beck, S. J., [411](#)
Becker, J. A. J., [213](#)
Becker, J. B., [113](#)
Becker, S., [537](#)
Beckett, L. A., [515](#)
Beckham, J., [457](#)
Bee, M. A., [82](#)
Beecham, G. W., [545](#)
Beek, T., [422](#)
Beethoven, L., [22](#)
Befort, K., [213](#)
Behnia, R., [444](#)
Bejder, L., [333](#)
Bekerian, D., [285](#)
Belichenko, P. V., [540](#)
Bell, C. C., [48](#)
Bell, M. R., [529](#), [537](#)
Bellucci, S., [539](#)
Benabid, A. L., [363](#)
Benali, K., [517](#)
Ben-Chaim, E., [297](#)
Bendor, D., [317](#)
Benjamin, M., [450](#)
Bennet, E., [44](#)
Bennett, D., [546](#)
Bennett, D. A., [515](#)
Bennett, M. R., [459](#)
Bennett, W., [336](#), [345](#)
Benning, S. D., [422](#)
Benninger, K. L., [491](#)
Benninger, W. B., [491](#)
Ben-Shakhar, G., [457](#)
Bentsi-Enchill, E., [422](#)
Berberian Ade, A., [456](#)
Berenbaum, S., [530](#)
Berg, S., [523](#), [526](#)

Bergen, M. T., [532](#)
Berger, T., [532](#)
Bergman, H., [362](#)
Berkowitz, L., [486](#)
Berman, A. J., [111](#)
Berman, K. F., [407](#)
Berman, M. C., [401](#)
Berman, M. G., [214](#)
Bernier, G., [322](#)
Bernstein, I. L., [168](#)
Berridge, K. C., [210–211](#), [218](#), [427](#)
Berry, C. J., [89](#)
Bertolino, A., [521](#)
Bertran, C., [314](#)
Besson, A., [433](#)
Best, T. K., [541](#)
Beyene, N. W., [175](#), [186](#)
Bhatnager, D. C., [77](#)
Bhupatkar, A., [336](#)
Bihrlé, A., [394](#)
Bimonte, H., [515](#)
Binkofski, F., [489](#)
Birbaumer, N., [158](#)
Bird, G., [494](#)
Birks, J., [70](#)
Birren, J., [517](#)
Bitter, I., [268](#)
Bjork, R. A., [285](#), [299](#), [336](#), [337](#)
Björk-Eriksson, T., [537](#)
Blair, H. T., [444](#)
Blakely, E., [191](#)
Blank, M., [540](#)
Blanton, S. R., [111](#)
Blascovich, J., [517](#)
Blendy, J., [165](#)
Blennow, K., [545](#)

Bliss, T. V., [63](#), [64](#)
Blundel, J., [545](#)
Boada, M., [545](#)
Boatman, P. R., [336](#)
Boche, D., [542](#)
Body, S., [201](#), [207](#)
Boesch, C., [333](#)
Bogart, D., [296](#)
Boisjoli, J. A., [186](#)
Boldogh, I., [545](#)
Bonanno, G. A., [457](#)
Bonardi, C., [245](#)
Bond, A. B., [89](#)
Bonheim, P., [82](#)
Bontempi, B., [314](#)
Boot, W. R., [351](#)
Born, J., [315](#), [316](#), [317](#)
Borszcz, G., [82](#)
Borton, D., [364](#)
Botez, M., [359](#)
Bouchard, T. J., Jr., [341](#)
Bourgeois, J.-P., [527](#)
Bourtchuladze, R., [165](#)
Bouton, M., [133](#), [169](#), [430](#), [458](#)
Bovbjerg, D. H., [169](#)
Bower, G. H., [143–144](#), [146](#), [231](#), [254](#)
Bowles, R. P., [516](#)
Boyes-Braem, P., [251](#)
Boyke, J., [355](#)
Boyle, R., [11](#)
Bozorgmehr, T., [532](#)
Braak, E., [543](#)
Braak, H., [543](#)
Bradbury, J. W., [482](#)
Bradshaw, C. M., [201](#), [207](#)
Brady, E., [344](#)

Braff, D. L., [83](#)
Braithwaite, D. W., [249](#)
Brakefield, T., [346](#)
Brambilla, P., [494](#)
Brand, M., [320](#)
Brandt, J., [338](#)
Bransford, J., [286](#), [288](#)
Braque, G., [251](#)
Brass, M., [493](#)
Brasure, M., [546](#)
Braun, C., [106](#)
Braun, D. A., [345](#)
Braver, T. S., [408](#)
Brehmer, Y., [334](#)
Breiter, H., [219](#)
Brembs, B., [210](#)
Bremner, J. D., [451](#)
Brewer, J., [306](#), [448](#)
Brickson, M., [517](#)
Bridgett, D., [525](#)
Brigham, J. C., [91](#)
Brinton, R., [532](#)
Bristol, A. S., [101](#)
Broccolini, A., [321](#)
Bronson, R., [540](#)
Brooks-Gunn, J., [507](#)
Broster, B. S., [80](#)
Brown, C. A., [485–486](#), [512](#)
Brown, F. L., [214](#)
Brown, G., [433](#)
Brown, J., [188](#), [494](#)
Brown, J. M., [322](#)
Brown, P., [339](#)
Brown, R., [436](#)
Brown, S., [480](#)
Brown, T., [525](#)

Bruce, C. J., [396](#)
Bruell, J., [125–126](#)
Brugge, K., [545](#)
Bruneau, R., [358](#)
Brunelli, M., [100](#)
Brunet-Bourgin, F., [441](#)
Bryan, R., [533](#)
Bryant, R., [419](#)
Bryson, S. E., [494](#)
Buccino, G., [489](#)
Buchanan, M., [376](#)
Buchanan, T., [450](#)
Buchel, C., [355](#)
Buchwald, J., [516](#)
Buckner, R. L., [306](#), [405](#)
Buffalo, E., [305](#), [312](#), [437](#)
Bugnyar, T., [477](#)
Buhl, D., [316](#)
Bullmore, E. T., [314](#)
Buonocore, M., [535](#)
Burchinal, M., [44](#)
Burd, G., [537](#)
Burgdorf, J., [428](#)
Burgess, N., [109](#)
Burke, S. N., [536](#)
Buros, J., [545](#)
Bursley, B., [545](#)
Burton, M. A., [545](#)
Burwitz, L., [341](#)
Bush, G. W., [215](#), [295](#)
Bushman, B. J., [252](#)
Bushman, E. J., [487](#)
Bushnell, I. W. R., [504](#)
Busnel, M.-C., [504](#)
Busso, D. S., [460](#)
Butki, B. D., [334](#)

Butler, A. C., [289](#), [290](#)
Butler, K., [356](#)
Butler, M., [546](#)
Butters, N., [338](#), [541](#)
Buzsáki, G., [310](#), [315](#), [316](#)
Byrne, J., [210](#)
Byrne, R. W., [475](#), [479](#)
Byrnes, M. L., [355](#)

C

Cabeza, R., [404](#), [405](#), [448](#), [536](#)
Cacucci, F., [109](#)
Cafini, F., [538](#)
Cahill, L., [55](#)
Cahill, L. R., [434](#), [435](#), [442](#), [447](#), [450](#), [515](#), [532](#)
Calhoun, P. S., [457](#)
Callaway, E. M., [537](#)
Callicot, J. H., [408](#)
Callicott, J., [521](#)
Campbell, G., [541](#)
Campese, V. D., [459](#)
Campolongo, P., [55](#)
Canestrari, R., Jr., [517](#)
Canli, T., [448](#)
Cannon, W., [423](#)
Cannon, W. B., [422](#)
Cantu, R. C., [319](#)
Capaldi, E., [308](#)
Capone, N. C., [512](#)
Caramanos, Z., [393](#)
Carder, D., [480](#)
Cardinal, R. N., [442](#)
Carell, S., [302](#), [304](#)
Carew, T. J., [82](#), [101](#), [162–164](#)
Carlander, B., [321](#)
Carlisimo, G., [539](#)
Carlson, A. A., [202](#)
Carlson, R. A., [330](#), [331](#)
Carmelli, D., [521](#), [526](#)
Carmena, J. M., [364](#)
Carnak, L., [474](#)
Carney, R. S., [541](#)

Carpenter, P., [388](#)
Carr, C., [243–244](#)
Carr, V. A., [304](#), [314](#)
Carra, R., [115](#)
Carrick, L. A., [407](#)
Carroll, K. M., [169](#)
Carroll, L. J., [319](#)
Carskadon, M. A., [536](#)
Carstensen, L., [518](#)
Carter, C. S., [408](#)
Carter, E., [250](#)
Carter, G., [243](#)
Carter, M., [320](#), [479](#)
Carvalho, P. F., [251](#)
Carver, M., [319](#)
Casale, M. S., [64](#)
Casal-Roscum, L., [519](#)
Casals, P., [517](#)
Caselli, R. J., [545](#)
Caspi, Y., [460](#)
Cassel, J., [308](#)
Cassel, S., [308](#)
Cassidy, J. D., [319](#)
Castellanos, F. X., [411](#)
Castellucci, V. F., [98](#), [100](#)
Catarino, A., [458](#)
Catmur, C., [490](#)
Cavalcanti-Ribeiro, P., [458](#)
Cavallina, C., [55](#)
Cavallini, A., [321](#)
Caverzasi, E., [321](#)
Caviness, V., [531](#)
Cebrian-Silla, A., [537](#)
Ceceli, A. O., [459](#)
Centerwall, B. S., [486](#)
Ceric, F., [218](#)

Cervone, K., [210](#), [211](#)
Cezanne, [251](#)
Chabris, C., [525](#)
Chace, P., [297](#)
Chadderton, H., [376](#)
Chakrabarti, L., [541](#)
Chan, D., [313](#)
Chang, J., [321](#), [322](#)
Chang, L. J., [214](#)
Chang, Y., [458](#)
Charles, S., [518](#)
Charman, T., [494](#)
Charness, N., [341](#)
Chauvel, P., [110](#), [441](#)
Chawla, M., [535](#)
Chee, M. W. L., [408](#)
Chen, B., [528](#)
Chen, C., [303](#)–[304](#)
Chen, L. Y., [64](#), [158](#)
Chen, M., [165](#)
Chen, S., [210](#)
Chen, Z., [316](#)
Cheney, D. L., [203](#)
Cheng, P., [298](#)
Chetelat, G., [361](#)
Chi, M., [514](#)
Chiba, A. A., [206](#)
Chitty, K. M., [459](#)
Choi, J., [537](#)
Choic, S. G., [202](#)
Choo, W. C., [408](#)
Chou, A., [545](#)
Chow, T. E., [56](#)
Christoff, K., [446](#)
Churchill, D. W., [494](#)
Ciaramelli, E., [207](#)

Cicchetti, P., [443](#)
Cincotta, C. M., [353](#)
Cioffi, D., [165](#)
Cipolotti, L., [313](#)
Cirelli, C., [315](#)
Ciszewski, A., [459](#), [460](#)
Clare, L., [243](#)
Clark, D., [435](#)
Clark, J., [266](#)
Clark, R. E., [110](#)
Clarke, G., [115](#)
Clayton, N., [283](#)–[284](#)
Clemenson, G. D., [524](#)
Clifford, R., [44](#)
Clinton, B., [302](#)
Clinton, H., [438](#)
Clouse, E., [361](#)
Clutton-Brock, T., [202](#)
Cohen, J. D., [408](#)
Cohen, L. G., [68](#)
Cohen, N. J., [168](#), [280](#), [297](#), [339](#)
Cohen, S. J., [109](#)
Cohen, T. E., [99](#)
Cohen-Bendahan, C., [530](#)
Cohen-Kettenis, P., [532](#)
Coleman, E. S., [113](#)
Collett, C., [277](#)
Collette, F., [316](#)
Collie, A., [541](#)
Collinger, J. L., [364](#)
Collins, A. G. E., [505](#)
Collins, D. M., [506](#)
Collins, K., [505](#)
Colobo, J., [77](#)
Colon, M., [283](#)
Colucci, P., [55](#)

Coluccia, D., [521](#)
Cominski, T. P., [532](#)
Comte, F., [321](#)
Condon, C. D., [262](#)
Conn, P., [349](#)
Connolly, C. I., [352](#)
Connor, R. C., [203](#), [333](#)
Conron, K. J., [113](#)
Contreras, M., [218](#)
Contreras-Vidal, J. L., [337](#)
Conturo, T., [407](#), [408](#)
Conway, M. A., [281–282](#)
Cook, M., [455](#), [475](#)
Cooke, S. F., [110](#)
Cooper, B. G., [109](#)
Cooper, J., [306](#)
Cooper, N. R., [358](#)
Cooper, R., [523](#)
Coppola, G., [77](#)
Corballis, M. C., [333](#)
Corbett, A. T., [336](#)
Corcoran, K., [458](#)
Cork, L. C., [517](#)
Corkin, S., [277](#), [278](#), [280](#), [308](#), [314](#), [338](#), [437](#), [541](#)
Cornstock, G., [486](#)
Coronado, V. G., [319](#)
Corsellia, J., [533](#)
Corter, J. E., [254](#)
Costa, R. M., [361](#), [364](#)
Costantini, E., [542](#)
Cottier, J. P., [489](#)
Coughlan, A., [310](#)
Courage, M., [507](#)
Courtine, G., [364](#)
Coutureau, E., [264](#), [267](#)
Cowan, N., [379](#)

Cowan, T., [437](#)
Cox, C., [175](#), [186](#)
Cox, C. L., [175](#), [187](#)
Craig, A. D., [214](#), [215](#)
Craik, F., [286](#)
Cramer, N. P., [541](#)
Crane, J., [393](#)
Cranney, J., [82](#)
Creso-Moyano, J., [266](#)
Crist, R., [364](#)
Crnic, L., [539](#)
Crook, M., [525](#)
Crystal, J. D., [283–284](#)
Csernansky, J., [407](#), [408](#)
Csicsvari, J., [315](#), [316](#)
Cuevas, J., [525](#)
Cuevas, K., [507](#)
Curio, E., [476](#)
Curran, T., [310](#)
Curtis, C. E., [395](#), [398](#)
Curtiss, S., [510](#)
Cusato, B., [152](#)
Custance, D. M., [472–473](#)
Cutler, N., [545](#)
Czaja, S., [517](#)
Czerlanis, C. M., [359](#)
Czurkó, A., [315](#), [316](#)

D

Dabholkar, A., [527](#)
Dahlstrom, K., [411](#)
Dahmer, J., [75–76](#)
Dale, A., [306](#)
Dale, A. M., [215](#), [219](#)
Dalgleish, T., [458](#)
Damasio, A. R., [310](#), [338](#), [392](#), [422](#), [442](#), [445](#)
Damasio, H., [218](#), [310](#), [338](#), [391](#), [442](#), [445](#)
Damon, M., [321](#)
Daneman, M., [388](#)
Daneshvar, D. H., [319](#)
Danion, J. M., [361](#)
Dannon, P. N., [297](#)
Danshevar, D. H., [319](#)
Darwin, C., [6](#), [15–17](#), [19](#), [26](#), [28](#), [31](#), [425](#)
Darwin, E., [15](#), [19](#)
Daselaar, S. M., [536](#)
Dash, P. K., [164](#)
Dashner, J., [321](#)
Daum, I., [158](#)
Dauta, A., [214](#)
Davachi, L., [287](#), [306](#)
Dávalos, A., [218](#)
Davids, K., [341](#)
Davidson, R., [451](#)
Davies, N., [352](#)
Davies, P., [545](#)
Davila, H., [546](#)
Davila-Ross, M., [428](#)
Davis, H., [82](#), [188](#)
Davis, M. I., [83](#), [361](#), [457](#), [459](#)
Davis, S. L., [459](#)

Davis-Dasilva, M., [474](#)
Davisson, M., [540](#)
Davoli, M., [220](#)
Daw, N., [206](#), [459](#)
Dawson, B. V., [473](#)
Dawson, G. R., [473](#)
Day, E. A., [336](#)
Day, J. J., [525](#)
Dayan, J., [321](#)
Deakin, J. F., [201](#), [207](#)
De Beaumont, L., [319](#)
de Bruijn, A., [488](#)
DeCasper, A., [503](#), [504](#)
DeGeneres, E., [324](#)
Deiber, M. P., [104](#)
Deisseroth, K., [444](#), [445](#)
DeKosky, S. T., [319](#)
De La Sayette, V., [321](#)
Del Boca, F. K., [252](#)
de Leon, M. J., [542](#), [543](#), [545](#)
Delespaul, P., [113](#)
Delgado, M. R., [436](#), [448](#)
Delis, D. C., [394](#), [545](#)
Della Marca, G., [321](#)
Delmaire, C., [214](#)
Del Negro, C., [490](#)
DeLong, M. R., [349](#), [362](#)
DeLuca, J., [310](#), [311](#)
De Maio, V. J., [319](#)
Demetriou, S., [352](#)
DeMeyer, M. K., [494](#)
Demiroeren, S., [515](#)
Demougin, P., [521](#)
DeMyer, W. E., [494](#)
Denenberg, V., [515](#)
Deng, W., [524](#)

Dennis, C. V., [536](#), [538](#)
Dennis, E. L., [459](#)
Dennis, N. A., [536](#)
Dennis, S. J., [507](#)
de Pellegrino, G., [207](#)
de Quervain, D., [450](#), [458](#), [521](#)
Dere, E., [283](#)
Derom, C., [113](#)
Derrfuss, J., [493](#)
Desai, P., [546](#)
Descartes, R., [9–10](#), [10–11](#), [14](#), [47](#), [331](#), [333](#)
Desgranges, B., [314](#), [517](#)
Desimone, R., [397](#)
Desmond, J. E., [306](#), [404](#)
Desmurget, M., [349](#)
De Souza Silva, M. A., [283](#)
D’Esposito, M., [380](#), [395](#), [398](#), [401](#), [402](#), [403](#), [405](#), [408](#), [491](#)
DeStefano, A. L., [545](#)
Destrieux, C., [489](#)
Detre, J. A., [408](#)
de Verbizier, D., [321](#)
Devitt, M., [80](#)
DeVoogd, T., [44](#)
de Vries, S., [456](#)
de Waal, F., [425](#)
de Waal, F. B. M., [506](#)
De Weerdt, W., [494](#)
Dewsbury, D. A., [79](#)
Dhara, M., [363](#)
Diamond, B., [310](#)
Diamond, M., [44](#)
Diana, R. A., [308](#)
Dias, B. G., [526](#)
Dichgans, J., [158](#)
Dickinson, A., [152](#), [283–284](#), [432](#)
Dickinson-Anson, H., [55](#), [537](#)

Diechmann, R., [206](#)
Diekelmann, S., [315](#), [316](#), [317](#)
Dierssen, M., [540](#)
Digdon, N., [454](#)
DiNardo, P., [455](#)
di Nemi, S. U., [494](#)
Dinse, H. R., [106](#), [355](#)
Dinsmoor, J. A., [431](#)
di Pellegrino, G., [489](#)
Ditman, T., [269](#)
Dittoni, S., [321](#)
Dixon, M. L., [446](#)
Dobbins, I. G., [404](#), [536](#)
Dolan, R., [203](#), [206](#)
Dolberg, O. T., [297](#)
Dolcos, F., [448](#)
Dollard, J., [470](#)
Domjan, M., [83](#), [84](#), [125–126](#), [152](#)
Donaldson, Z. R., [427](#)
Dong, Q., [303–304](#)
Donnerstein, E., [486](#)
Doorn, C., [532](#)
Dostrovsky, J., [107](#)
Dougherty, S., [525](#)
Down, J. L., [539](#)
Downey, J. E., [364](#)
Dowsey-Limousin, P., [363](#)
Doyon, J., [331](#), [359](#)
Draganova, R., [503](#)
Dressler, M., [290](#)
Driemeyer, J., [355](#)
Dr. Seuss, [503](#)
Duara, R., [543](#)
Dudai, Y., [296](#), [298](#), [313](#)
Duff, K., [545](#)
Duffy, L., [243](#)

Dumitriu, D., [533](#), [535](#)
Duncan, C., [297](#)
Duncan, J., [393](#)
Duncan, K., [306](#)
Duning, K., [521](#)
Dunlosky, J., [299](#)
Dunsmoor, J. E., [459](#)
Durstun, S., [412](#)
Dutton, D. G., [423](#)
Duys, A., [460](#)
Dwyer, J., [296](#)
Dymond, S., [431](#)
Dzokoto, V., [422](#)

E

Eacott, M. J., [284](#)
Eals, M., [515](#)
Easton, A., [284](#)
Eaton, B. A., [536](#)
Eayrs, C. B., [243](#)
Ebbinghaus, H., [19–21](#), [291](#)
Eckerman, C., [505](#), [526](#)
Eckerson, J. M., [344](#)
Eckstrand, K., [403](#)
Edeline, J-M., [490](#)
Edgin, J., [540](#)
Edison, T., [139](#)
Egan, M. F., [408](#), [521](#)
Eich, E., [435](#)
Eichenbaum, H., [88](#), [269](#), [282](#), [298](#), [308](#), [314](#), [338](#)
Einstein, A., [23](#)
Eisenberg, N., [419](#)
Eisenberger, N. I., [214](#)
Eisenstein, D., [82](#)
Eisenstein, E. M., [82](#)
Ekman, P., [419](#), [422](#)
Elbert, T., [111](#), [355](#)
Ellgring, H., [444](#), [456](#)
Elliott, L., [322](#)
Elliott, M. A., [531](#)
Ellis, C. B., [458](#)
Ellis, T., [183](#)
Ellsworth, D. W., [166](#)
Elster, A., [533](#)
Ely, T., [448](#)
Emmelkamp, P., [456](#)
Emslie, H., [393](#)

Engel, S. A., [314](#)
Engle, R. W., [352](#)
Ensminger, J., [184](#)
Epp, J. R., [537](#)
Epstein, C. J., [540](#)
Epstein, D., [220](#)
Erb, M., [106](#)
Erbes, C. R., [461](#)
Erickson, C. A., [397](#)
Erickson, K. I., [351](#)
Erickson, M. A., [401](#)
Ericsson, K. A., [290](#), [340](#)
Eriksson, P., [537](#)
Ermita, B., [310](#)
Ernst, U., [476](#)
Erós-Samyai, M., [457](#)
Ertman, N., [532](#)
Eschaliér, A., [433](#)
Escorihuela, R., [540](#)
Eslinger, P. J., [310](#), [392](#)
Esseily, R., [502](#)
Estes, W. K., [24–26](#), [28](#), [126–127](#), [235](#)
Eswaran, H., [503](#)
Eustache, F., [321](#), [352](#), [517](#)
Evans, A. C., [394](#), [399](#), [400](#)
Evans, A. H., [62](#)
Evans, D., [546](#)
Evans, D. A., [515](#)
Evans, J. G., [70](#)
Evans, S., [283–284](#)
Everaerd, W., [80](#)
Everitt, B. J., [216](#), [442](#)
Exner, C., [339](#)

F

Faber, T. L., [411](#)
Fadiga, L., [489](#)
Fagard, J., [502](#)
Fahlström, A., [517](#)
Fahrback, S., [308](#)
Fallon, J., [447](#)
Fallon, J. H., [55](#)
Fan, X., [535](#)
Fanselow, M., [429](#), [442](#)
Farah, M. J., [405](#)
Farina, L. M., [321](#)
Farkas, M., [268](#), [270](#)
Farrell, C., [324](#)
Featherstone, R., [205](#)
Fechner, G., [19](#)
Feeling, N., [457](#)
Feeney, M. C., [283](#)
Fein, D., [494](#)
Fendt, [429](#)
Feng, G., [528](#)
Ferguson, C. J., [189](#), [487](#)
Fernald, R., [508](#)
Fernandez, F., [540](#)
Fernández, G., [290](#), [298](#)
Fernandez de Molina, A., [441](#)
Fernandez-Teruel, A., [540](#)
Fernel, E., [411](#)
Ferrari, M., [334](#)
Ferri, A., [220](#)
Ferris, S., [544](#)
Fialip, J., [433](#)
Fields, R. D., [42](#)

Filion, D. L., [338](#)
Finch, C., [545](#)
Fine, I., [104](#)
Fink, H. A., [546](#)
Finkel, D., [523](#)
Firpi, H. A., [542](#)
Fischman, M., [212](#)
Fisher, A. E., [79](#)
Fisher, E. M. C., [539](#)
Fisher, P. A., [113](#)
Fitts, P., [340](#), [353](#)
Fitzpatrick, A. L., [545](#)
Fivush, R., [507](#)
Flaherty, C. F., [185](#)
Fleck, M. S., [536](#)
Fletcher, P. C., [314](#)
Flinn, M. V., [427](#)
Floel, A., [68](#)
Florer, F., [436](#)
Flores, C., [529](#), [537](#)
Flórez, J., [540](#), [541](#), [546](#)
Flor-García, M., [538](#)
Flourens, P., [59](#)
Flynn, J., [209](#)
Fogarty, S., [435](#)
Fogassi, L., [489](#)
Foley, H., [404](#)
Foltin, R., [212](#)
Fones, C. S. L., [408](#)
Fong, G., [210](#)
Font-Mayolas, S., [218](#)
Forbes, N. F., [407](#)
Ford, J. K., [346](#)
Forssberg, H., [411](#)
Foss, B. M., [473](#)
Fowler, J., [411](#)

Fox, N., [313](#), [524](#)
Fox, P. W., [341](#)
Fox, R., [444](#)
Foxe, J. J., [215](#)
Foy, M., [532](#)
Frackowiak, R. S. J., [44](#), [314](#), [359](#)
Francis, P., [62](#)
Frank, M. J., [506](#)
Franks, J., [288](#)
Franz, T. M., [206](#)
Freedman, M., [322](#)
Freeman, J., [516](#)
Freer, C., [393](#)
Frenda, S. J., [295](#)
Frenguelli, B., [165](#)
Fried, I., [55](#)
Fried, L., [302](#)
Friedman, D., [519](#)
Friedman, S., [457](#)
Friesen, W., [419](#)
Frigerio, C. S., [543](#)
Fristen, K., [206](#)
Friston, K. J., [314](#), [359](#)
Frisullo, G., [321](#)
Frith, C. D., [44](#), [359](#)
Frommen, J. G., [203](#)
Fry, A., [201](#)
Fuchs, S., [316](#)
Fudin, R., [525](#)
Fujii, T., [322](#)
Fulton, J. J., [457](#)
Funahashi, S., [396](#), [401](#)
Furedy, J., [515](#)
Fuster, J. M., [395](#), [401](#)
Futterman, A. D., [169](#)

G

Gabrieli, I. D., [404](#), [406](#)

Gabrieli, J., [306](#)

Gabrieli, J. D. E., [280](#), [306](#), [338](#), [353](#), [360](#), [448](#), [541](#)

Gabrieli, S., [306](#)

Gadagkar, R., [202](#)

Gadian, D. G., [44](#)

Gaffan, D., [307](#)

Gage, F. H., [310](#), [524](#), [537](#)

Gaither, G., [80](#)

Galani, R., [308](#)

Galbicka, G., [183](#)

Galdzicki, Z., [541](#)

Galea, A., [514](#)

Galea, L. A., [537](#)

Galea, S., [457](#)

Galef, B. G., Jr., [484](#), [485](#)

Gall, C. M., [64](#), [70](#)

Gall, F. J., [39–40](#), [43](#)

Gallagher, M., [206](#), [441](#), [533](#), [535](#)

Gallese, V., [489](#)

Galli, G., [286](#)

Gallins, P. J., [545](#)

Galván, A., [530](#)

Galvao-de Almeida, A., [456](#)

Gamer, M., [437](#)

Gandy, S., [542](#)

Gangitano, M., [489](#)

Ganguly, K., [364](#)

Garavan, H., [215](#)

Garcia, J., [151–152](#)

Garcia, R., [442](#), [448–449](#)

Gardner-Medwin, A., [63](#), [65](#)

Garner, C. C., [540](#)
Garry, J., [294–295](#)
Gaser, C., [355](#)
Gatchel, R., [81](#)
Gathercole, S., [513](#)
Gavornik, J. P., [110](#)
Gaznick, N., [218](#)
Geary, D. C., [427](#)
Geda, Y. E., [545](#)
Gede, [525](#)
Geen, R. G., [486](#)
Gentes, E. L., [113](#)
George, A., [542](#)
Georgopoulos, A. P., [54](#)
Gerler, D., [299](#)
Gerloff, C., [68](#)
Gerrard, J. L., [536](#)
Gershberg, F. B., [404](#)
Gershoff, E. T., [189](#)
Geschwind, N., [113](#)
Getty, T., [203](#)
Geyer, M. A., [77](#), [83](#)
Ghirardi, M., [165](#)
Ghosh, J. G., [545](#)
Giang, D. W., [168](#)
Gibson, E. J., [89](#), [90](#), [91](#)
Gibson, J. J., [90](#)
Gil, S., [460](#)
Gilberg, C. G., [411](#)
Gilbertson, M. W., [459](#), [460](#)
Gilchrist, A., [495](#)
Gilewski, T., [169](#)
Gilliam, K. S., [113](#)
Gilman, S. E., [113](#)
Gilmore, J. H., [529](#)
Gilmore, R. O., [330](#), [331](#)

Giret, N., [490](#)
Giurfa, M., [202](#)
Glantz, L. A., [529](#)
Glanzman, D. L., [77](#)
Glass, A. L., [291](#)
Glass, R., [297](#)
Glauthier, S., [167](#)
Globus, A., [44](#)
Glover, G. H., [306](#), [404](#)
Gluck, M. A., [144–145](#), [147](#), [159](#), [161](#), [231](#), [254](#), [265–266](#), [270](#), [310](#), [354](#), [544](#)
Godde, B., [106](#)
Godden, D., [288](#)
Godfrey, M., [539](#)
Goelz, R., [503](#)
Goff, D., [267](#)
Goff, L., [295](#)
Gogtay, N., [403](#)
Gold, A. L., [458](#)
Gold, J. M., [267](#)
Gold, P., [449](#)
Goldberg, T., [521](#)
Goldberg, T. E., [408](#)
Goldberg, W., [302](#)
Goldman, D., [408](#)
Goldman, S., [537](#)
Goldman-Rakic, P. S., [391](#), [395–396](#), [401](#), [529](#)
Goldschmidt, R., [308](#)
Goldstein, J., [531](#)
Goldstein, M. B., [461](#)
Goldstone, R. L., [249](#), [251](#)
Golinkoff, R. M., [525](#)
Golm, D., [524](#)
Golomb, J., [544](#)
Gomez, J. C., [472–473](#)
Gonzalez, A., [277](#)
Gonzalez-Martinez, V., [321](#)

Good, C. D., [44](#)
Good, M., [264](#), [267](#)
Goodall, J., [333](#)
Goodman, A. D., [168](#)
Goodwin, J. E., [344](#)
Goodwyn, J. E., [512](#)
Goodwyn, S. W., [512](#)
Gooren, L., [532](#)
Gorbachov, M., [308](#)
Gordon, A., [419](#)
Gordon, E., [446](#)
Gordon, R., [488](#)
Górecka-Bruzda, A., [184](#)
Gorelick, D., [216](#), [221](#)
Gormezano, I., [127](#), [133](#)
Gottschalk, W., [521](#)
Goudsmit, E., [532](#)
Gould, E., [537](#)
Govindarajan, A., [445](#)
Goyal, V., [363](#)
Grace, A., [443](#)
Graf, P., [88](#), [280](#), [517](#)
Graff-Radford, N., [310](#)
Grafman, J., [104](#)
Grafton, S. T., [349](#), [411](#), [448](#)
Graham, K. S., [314](#)
Grajski, K. A., [355](#)
Granier-Deferre, C., [504](#)
Grant, J. E., [216](#), [221](#)
Gras, M. E., [218](#)
Grau, A., [218](#)
Gray, J. H., [525](#)
Gray, R. D., [333](#), [477](#)
Gray, W. D., [251](#)
Graybiel, A. M., [339](#), [349](#), [352](#), [353](#)
Graziano, A., [359](#)

Graziano, M., [537](#)
Grea, H., [349](#)
Green, A. E., [543](#)
Green, C. S., [352](#)
Green, J. G., [460](#)
Green, L., [201](#)
Green, R., [532](#)
Greenberg, D. L., [437](#)
Greene, C. M., [457](#)
Greengard, P., [545](#)
Greenough, W. T., [44](#), [359](#)
Greenwald, A. G., [91](#)
Greicius, M., [210](#)
Greicius, M. D., [290](#)
Greig, M. T., [543](#)
Greve, D., [215](#)
Griffin, A. S., [202](#)
Grill-Spector, K., [304](#)
Groccia-Ellison, M., [516](#)
Grogan-Kaylor, A., [189](#)
Gross, A. M., [487](#)
Gross, C., [537](#)
Grossman, M., [408](#)
Groves, P. M., [84–85](#)
Growden, J. H., [280](#), [338](#), [541](#)
Grunhaus, L., [297](#)
Gudnason, V., [545](#)
Guilleminault, C., [536](#)
Guillery-Girard, B., [321](#)
Guillory, J. E., [475](#)
Gulley, J. M., [529](#)
Gundersen, H., [531](#)
Gunning-Dixon, F., [533](#)
Gurvits, T. V., [460](#)
Gustafsson, P., [411](#)
Guttman, H., [229–231](#), [232](#), [233](#), [240](#)

Guzy, L., [455](#)

H

Haag, R., [186](#)
Hackenberg, T. D., [186](#)
Haglund, K., [277](#)
Hahn, M. E., [337](#)
Haier, R. J., [447](#)
Hair, L. P., [457](#)
Halgren, E., [441](#)
Hall, D. J., [200](#)
Hall, G., [91](#), [245](#)
Hall, T., [533](#)
Hallett, M., [104](#)
Hallion, L. S., [113](#)
Hamanaka, H., [444](#)
Hamann, S., [448](#)
Hambrick, D. Z., [352](#)
Hamer, R. M., [529](#)
Hamilton, A. F. D. C., [494](#)
Hamilton, W. D., [202](#), [203](#)
Hammond, K., [517](#)
Han, I. W., [321](#)
Hancock, J. T., [475](#)
Handwerk, B., [290](#)
Hanewinkel, R., [488](#)
Haney, M., [212](#)
Hanley-Dunn, P., [517](#)
Hannan, A. J., [546](#)
Hanson, C. A., [491](#)
Hao, J., [533](#), [535](#)
Hara, J., [535](#)
Hara, Y., [533](#)
Harand, C., [314](#)
Hardan, A., [494](#)

Hardy, J., [317](#)
Hari, R., [495](#)
Harkness, K. L., [113](#)
Harlow, H. F., [345](#)
Harnden, T., [438](#)
Harris, B., [454](#), [540](#)
Harris, C., [454](#)
Harris, J. E., [518](#)
Harris, K., [295](#)
Harrison, G., [294](#), [313](#)
Harrison, J., [516](#)
Harrison, R. H., [239–241](#)
Harsch, N., [437](#)
Hart, B., [333](#)
Hart, N., [545](#)
Hartman, B. K., [220](#)
Hartmann, R., [115](#)
Hasselmo, M. E., [310](#)
Hastings, G., [488](#)
Hatcher, M. B., [460](#)
Hatze, H., [335](#)
Hawkins, R. D., [99](#), [109](#), [162–164](#)
Haxby, J., [545](#)
Haydar, T. F., [540](#)
Hayden, E. P., [113](#)
Hayes, C., [473](#), [480](#)
Hayes, J. P., [459](#)
Hayes, K. J., [473](#)
Hayes, N., [537](#)
Hayes, S. M., [536](#)
Hayne, H., [505](#), [507](#)
Hayward, M. D., [213](#)
Hayworth, R., [540](#)
Head, D., [533](#)
Hebb, D., [30](#), [45–46](#)
Heckers, S., [267](#)

Heid, S., [115](#)
Heindel, W., [541](#)
Heithaus, M. R., [333](#)
Helgeson, V. S., [426](#)
Hellmann-Regen, J., [542](#)
Helmes, E., [322](#)
Hemsley, D., [435](#)
Henderson, S., [80](#)
Hensch, T. K., [104](#)
Henson, R. N. A., [89](#), [287](#)
Hepper, P., [503](#)
Herbert, J., [505](#), [526](#)
Herder, S. P., [165](#)
Herman, L. M., [284](#), [333](#), [479](#)
Herrmann, M., [456](#)
Herrnstein, R., [198](#)
Hershberger, S. L., [341](#)
Hesten, L., [545](#)
Hester, R., [214](#), [215](#)
Heston, C., [541](#)
Hetrick, W. P., [144](#)
Heuer, F., [434](#)
Heyes, C., [490](#)
Heyes, C. M., [476](#), [477](#), [494](#)
Hikosaka, O., [213](#)
Hilario, M. R. F., [361](#)
Hilgard, E., [127](#)
Hill, L., [545](#)
Hillegaart, V., [352](#)
Hinde, R. A., [482](#)
Hines, M., [532](#)
Hingtgen, J. N., [494](#)
Hinkel, D., [184](#)
Hinshaw, S., [491](#)
Hinson, R. E., [167](#)
Hirase, H., [315](#), [316](#)

Hiroto, D., [433](#)
Hirsh, R., [350](#)
Hirsh-Pasek, J. M., [525](#)
Hirst, W., [436](#), [437](#)
Hitch, G., [374](#)
Hitchcock, A., [321](#)
Ho, M. Y., [201](#), [207](#)
Hobel, Z., [544](#)
Hobson, J. A., [346](#)
Hochberg, L. R., [364](#)
Hochner, B., [164](#)
Hock, C., [450](#), [521](#)
Hodges, J. R., [243](#), [314](#), [320](#)
Hodzic, A., [106](#)
Hoefnagel-Höhle, H., [512](#), [513](#)
Hof, P., [533](#)
Hoffman, J. M., [349](#), [411](#)
Hoffman, K. L., [106](#)
Hoffman, S. J., [334](#)
Hoffmann, M., [456](#)
Hoffman-Snyder, C. R., [545](#)
Hogg, T. M., [358](#)
Holding, D. H., [334](#)
Holloway, J. L., [532](#)
Holloway, T., [361](#)
Holm, L., [319](#)
Holmes, C., [542](#)
Holmes, J., [215](#)
Holmes, W., [352](#)
Holzhaidner, J. C., [477](#)
Holzman, P. S., [269](#), [407](#)
Hommre, D., [210](#)
Honey, R. C., [245](#), [264](#), [267](#)
Honzik, C. H., [93](#)
Hooper, S., [479](#)
Hopkins, R., [312](#)

Hopkins, R. O., [266](#)
Hopkins, V., [542](#)
Hoplight, B., [515](#)
Hopper, L. M., [475](#)
Horn, G., [43](#)
Hornak, J., [307](#)
Horner, V., [472](#)
Horowitz, A. C., [474](#)
Horton, N., [531](#)
Howard, R. W., [340](#), [341](#)
Howard-Jones, P., [352](#)
Howe, M., [507](#)
Hu, D., [349](#)
Hu, F. B., [457](#), [460](#)
Hubel, D., [509](#), [528](#)
Huber, B. R., [319](#)
Hubert, V., [361](#)
Hudson Kam, C. L., [512](#)
Huentelman, M. J., [521](#)
Huesmann, L. R., [486](#)
Huettel, S. A., [536](#)
Hull, C., [23–24](#), [25](#), [29](#), [127](#), [135](#), [185](#)
Hulme, C., [376](#)
Hulsbosch, A., [456](#)
Hulse, S., [536](#)
Hunt, G. R., [333](#), [477](#)
Husain, G., [525](#)
Huston, J. P., [284](#)
Huttenlocher, P., [527](#)
Hwang, K. S., [543](#)
Hyde, J. S., [514](#), [515](#)
Hyde, L., [515](#)
Hyman, B., [277](#)

I

Ibanez, V., [104](#)

Ihori, N., [487](#)

Ikemoto, S., [217](#)

Ikram, M. A., [545](#)

Immelman, K., [482](#)

Imuta, K., [507](#)

Ingalhalikar, M., [531](#)

Innocence Project, [296](#)

Insausti, R., [305](#)

Insel, T., [508](#)

International Human Genome Sequencing Consortium, [520](#)

Irle, E., [339](#)

Irons, G., [454](#)

Irving, N., [540](#)

Irwin, A. R., [487](#)

Isaac, J. T., [541](#)

Isbell, L. A., [203](#)

Ivkovich, D., [505](#)

Iwata, J., [443](#)

J

Jack, C. R., Jr., [544](#)

Jack, L., [526](#)

Jackowski, A. P., [456](#)

Jacobs, B., [531](#)

Jacobs, N., [113](#)

Jacobsen, C., [377](#), [394](#)

Jacobsen, P. B., [169](#)

Jacquey, L., [502](#)

Jaffee, M. S., [319](#)

Jagust, W. J., [544](#)

James, J., [516](#)

James, W., [3–4](#), [3f](#), [6](#), [19](#), [28](#), [29](#), [30](#), [43](#), [45](#), [87](#), [243–244](#), [339](#), [372](#), [421–422](#), [422–423](#)

Janicki, D., [426](#)

Janik, V., [479](#)

Janowsky, J. S., [393](#)

Janssen, W. G. M., [533](#), [535](#)

Jarosiewicz, B., [364](#)

Jarrard, L., [308](#)

Jarskog, L. F., [529](#)

Jauffret, C., [214](#)

Javadi, A. H., [298](#)

Jefferson, T., [11](#)

Jeffries, M., [480](#)

Jenike, M. A., [215](#)

Jenkins, H. M., [239–241](#), [242](#)

Jenkins, K. G., [321](#)

Jenkins, P. F., [483](#)

Jenkins, W. M., [355](#), [356](#)

Jethanamest, D., [115](#)

Jezequel, J., [545](#)

Jezierski, T., [184](#)

Jezzard, P., [357–358](#)

Ji, D., [316](#)
Jin, D. Z., [349](#)
Jin, X., [364](#)
Jin, Y., [393](#)
Jog, M. S., [352](#)
Joh, A. S., [345](#)
Johansen, J. P., [444](#)
Johansen-Berg, H., [42](#)
Johansson, B., [523](#), [526](#)
Johnson, D. C., [461](#)
Johnson, D. M., [251](#)
Johnson, G., [542](#)
Johnson, J. D., [486](#)
Johnson, K., [277](#)
Johnson, M. H., [286](#), [411](#), [509](#), [512](#)
Johnson, R., [393](#)
Johnsrude, I. S., [44](#)
Johnston, J., [432](#)
Jones, C., [352](#)
Jones, G. V., [285](#)
Jones, J. D., [113](#)
Jones, R. S., [243](#)
Jonides, J., [379](#), [401](#)
Jonsdottir, I., [545](#)
Jonsson, P. V., [545](#)
Jonsson, T., [545](#)
Jordan, N. R., [202](#)
Josephson, W. L., [487](#)
Joslyn, S. L., [292](#)
Jun, G., [545](#)
Jungreis, C., [533](#)
Jurica, P. J., [404](#)
Jussim, L., [252](#), [254](#)
Justo, D., [214](#)

K

Kabani, N. J., [403](#)
Kabat-Zinn, J., [451](#)
Kagerer, F. A., [337](#)
Kahn, D. M., [104](#)
Kahneman, D., [219](#)
Kalish, N., [229–231](#), [232](#), [233](#), [240](#)
Kamil, A. C., [89](#)
Kamin, L., [137–138](#), [140–142](#)
Kandel, E. R., [97–98](#), [99](#), [100](#), [162–164](#)
Kane, M. J., [352](#)
Kane, R. L., [546](#)
Kanit, L., [515](#)
Kanner, L., [494](#)
Kaplan, E. S., [110](#)
Kaplan, S. W., [99](#)
Kapp, B., [441](#)
Kappenman, E. S., [144](#)
Kapur, N., [290](#), [310](#), [314](#), [320](#), [321](#)
Karim, A. A., [106](#)
Karl, A., [460](#)
Karl, K. A., [165](#)
Karlen, S. J., [104](#)
Karni, A., [357–358](#)
Kart-Tecke, E., [284](#)
Kasai, K., [459](#), [460](#)
Kassell, N., [310](#)
Kassin, S. M., [296](#)
Kaszniak, A. W., [310](#)
Kaufman, J., [525](#)
Kaufmann, J., [533](#), [535](#)
Kazdin, A., [455](#)
Kazwala, R., [187](#)

Keater, D., [393](#)
Keator, D., [447](#)
Kehoe, E. J., [127](#), [247](#)
Kelche, C., [308](#)
Kelemen, O., [266](#), [268](#)
Kelley, K. W., [537](#)
Kelly, J. L., [359](#)
Kelly, V. E., [337](#)
Kemper, T., [535](#)
Kempermann, G., [537](#)
Kenis, G., [113](#)
Kennedy, D., [531](#)
Kennedy, J. F., [308](#), [436](#)
Kennedy, M., [524](#)
Kensinger, E. A., [308](#), [437](#), [518](#)
Kéri, S., [268](#), [270](#)
Kerner, J., [296](#)
Kerns, K. A., [493](#)
Kersten, A., [251](#)
Khaw, M. W., [214](#)
Kiefer-Schmid, I., [503](#)
Kieffer, B. L., [213](#)
Kiernan, P. T., [319](#)
Kihlstrom, J., [321](#)
Kikuchi, H., [322](#)
Kilburn, J., [487](#)
Kilcross, S., [442](#)
Kilgard, M., [263](#)
Killcross, A. S., [264](#), [267](#)
Kilpatrick, L., [435](#)
Kilts, C. D., [411](#), [448](#)
Kim, J., [160](#)
Kim, J. K., [158](#)
Kim, J. S., [321](#), [437](#)
Kim, S. W., [220](#)
Kim, Y. K., [321](#)

Kimura, D., [514](#)
King, D., [133](#)
King, J. R., [482](#)
King, M. L., Jr., [436](#)
King George III of England, [15](#)
Kirchmayer, U., [220](#)
Kleim, J. A., [358](#)
Klein, B., [175](#)
Klengel, T., [526](#)
Kleschevnikov, A. M., [540](#)
Klingberg, T., [411](#)
Klinke, R., [115](#)
Kluger, A., [544](#)
Knapp, H. D., [111](#)
Knight, R. T., [404](#)
Knights, N., [524](#)
Knopman, D. S., [544](#)
Knott, G., [528](#)
Knowles, E. D., [295](#)
Knowlton, B. J., [280](#), [314](#), [338](#), [354](#)
Knutson, B., [210](#)
Kobre, K., [185](#)
Koch, C., [302](#)
Koechlin, E., [403](#), [408](#)
Koedinger, K. R., [336](#)
Koelling, R. A., [151–152](#)
Koenen, K. C., [113](#)
Köhler, S., [288](#)
Kojima, M., [521](#)
Kolachana, B. S., [408](#), [521](#)
Kolb, B., [446–447](#)
Kollaras-Mitsinikos, L., [460](#)
Koltai, E., [545](#)
Komorowski, R. W., [110](#)
Konrad, B. N., [290](#)
Kopelman, M. D., [321](#)

Koralek, A. C., [364](#)
Kordus, R. N., [334](#)
Koriat, A., [299](#)
Kornell, N., [299](#)
Korr, H., [537](#)
Koschack, J., [339](#)
Kosten, T., [218](#)
Kotrschal, K., [477](#)
Koukounas, E., [80](#)
Kouneiher, F., [403](#), [408](#)
Koutstaal, W., [306](#), [406](#)
Kowollik, V., [336](#)
Kozak, M. J., [433](#)
Kraft, T. L., [422](#)
Krakauer, J. W., [68](#), [339](#)
Kral, A., [115](#)
Kramer, A. D. I., [475](#)
Kramer, A. F., [351](#)
Krampe, R., [340](#)
Krank, M. D., [167](#), [168](#)
Krasnegor, N., [505](#)
Kraus, J., [319](#)
Kravitz, L., [91](#)
Kremerskothen, J., [521](#)
Krendl, A. C., [437](#)
Kreppner, J., [524](#)
Kriegeskorte, N., [303](#)
Krieman, G., [302](#)
Krijn, M., [456](#)
Kril, J. J., [536](#), [538](#)
Krimer, L. S., [529](#)
Kring, A., [419](#)
Kristof, N., [175](#)
Kritchevsky, M., [320](#), [321](#), [322](#), [393](#)
Kroes, M. C., [298](#)
Kross, E., [214](#)

Krubitzer, L., [104](#)
Kruggel, F., [55](#)
Krupa, D. J., [158](#), [160](#)
Kruschke, J. K., [144](#)
Krutzen, M., [333](#)
Kruyt, W., [94–95](#)
Kubota, K., [395](#)
Kubota, Y., [349](#), [352](#)
Kudrow, L., [302](#)
Kuhl, B., [306](#)
Kuhl, P. K., [479](#), [511](#)
Kuhn, H., [537](#)
Kujala, Y., [106](#)
Kulik, J., [436](#)
Kumar, N., [314](#)
Kumsta, R., [524](#)
Kupfermann, I., [98](#)
Küpper, C. S., [458](#)
Kushnir, T., [506](#)
Kwak, Y. T., [321](#)
Kwon, H., [514](#)
Ky, K., [525](#)

L

- Laan, E., [80](#)
- LaBar, K. S., [421](#), [426](#), [448](#)
- Labrecque, R., [322](#)
- Ladd, G. W., [471](#)
- Laforce, R., Jr., [359](#)
- Lagopoulos, J., [446](#), [459](#)
- Lah, J. J., [542](#)
- La Joie, R., [314](#)
- Lakhani, Y. S., [532](#)
- Laland, K. N., [485–486](#)
- Lalonde, R., [359](#)
- Lambe, E. K., [529](#)
- Lambeth, S. P., [475](#)
- Land, D., [288](#)
- Landeau, B., [314](#), [361](#)
- Landis, L. S., [517](#)
- Lang, P. J., [83](#)
- Lange, C., [421–422](#)
- Langlois, J. A., [319](#)
- Laplagne, D. A., [537](#)
- Larocque, K. F., [304](#)
- Lashley, K., [30](#), [59](#), [106](#), [182](#), [278](#), [304](#), [311](#)
- Lasko, N. B., [457](#), [458](#), [460](#)
- Laurent, H. K., [113](#)
- Laureys, S., [316](#)
- Lavond, D. G., [157](#)
- Lawrie, S. M., [407](#)
- Leaton, R., [82](#)
- Lebedev, M. A., [364](#)
- Lebrecht, S., [91](#)
- Lecanuet, J.-P., [504](#)
- LeDoux, J. E., [298](#), [421](#), [422](#), [441](#), [442](#), [443](#), [444](#), [445](#), [459](#)

Lee, A. K., [315](#)
Lee, J., [537](#)
Lee, N. R., [539](#)
Lee, T. D., [334](#)
Lees, A. J., [62](#)
Leggio, M. G., [359](#)
Lehericy, S., [362](#)
Lehman, A., [340](#)
Leibniz, G., [11](#)
Leighton, J., [494](#)
Lembeisis, E., [525](#)
Le Merrer, J., [213](#)
Lemieux, S. K., [158](#)
Lenck-Santini, P. P., [108](#)
Lennon, R., [419](#)
Lenroot, R., [403](#)
Leonard, G., [393](#)
LePort, A. K. R., [55](#)
Lerch, J. P., [403](#)
Levenson, R., [420](#)
Lever, C., [109](#)
Levesque, L., [344](#)
Levine, B., [314](#)
Levinson, S., [454](#)
Levy, B. J., [457](#)
Levy, D. L., [269](#)
Lewis, C., [339](#)
Lewis, D. J., [298](#)
Lewis, L. B., [104](#)
Lewis, M. C., [507](#)
Lewis, R. L., [401](#)
Lewis, T., [509](#)
Lewis-Peacock, J. A., [401](#)
Li, S. C., [334](#)
Liberzon, I., [440](#), [456](#), [457](#)
Liddle, P. F., [359](#)

Lieberman, J. A., [529](#)
Lieberman, M. D., [214](#)
Light, L., [517](#)
Likhtik, E., [448](#)
Lim, S-S., [42](#)
Lin, Y.-G., [450](#)
Lincoln, A., [285](#)
Linden, W., [455](#)
Lindenberger, U., [334](#)
Lindsay, S., [294–295](#)
Lindy, J., [209](#)
Ling, H., [319](#)
Linton, M., [281](#)
Linz, D., [486](#)
Lipka, J., [456](#)
Lipsett, L., [505](#)
Lipsitt, L., [185](#)
Lisman, J., [401](#)
Lit, L., [184](#)
Litchfield, C. A., [472](#)
Little, A. C., [505](#)
Liu, J., [334](#)
Liu, R. T., [113](#)
Liu, X., [445](#)
Llorens-Martín, M., [538](#), [545](#), [546](#)
Lockard, J. M., [158](#)
Locke, J., [10–11](#), [12](#), [14](#)
Lockett, G. A., [525](#), [526](#)
Lockhart, R., [286](#)
Loewenstein, D. A., [543](#)
Loftus, E. F., [294](#), [295](#)
Logan, C., [533](#)
Logan, J., [411](#)
Logothetis, N. K., [106](#)
Logue, M. W., [459](#)
Lombardi, G., [537](#)

Lømo, T., [63](#), [64](#)
Long, J. D., II, [364](#)
Longman, D., [336](#)
Lopez-Duran, N. L., [113](#)
Lorenz, K., [508](#)
Lott, D., [181](#)
Lou, W., [533](#), [535](#)
Louzada, F. M., [317](#)
Lovaas, O., [183](#)
Lovallo, W., [450](#)
Lovibond, P. F., [432](#)
Low, J., [525](#)
Low, M. J., [213](#)
Lozada, M., [81](#)
Lu, A., [391](#)
Lu, B., [521](#)
Lu, S., [545](#)
Lu, Z., [303](#)–[304](#)
Lublin, A. L., [542](#)
Lubow, R., [147](#), [265](#)
Lucas, E., [384](#)
Luck, S. J., [379](#)
Ludy, M-J., [82](#)
Lukashin, A., [54](#)
Lumbreras, M., [540](#)
Luria, A. R., [492](#)–[493](#)
Lüscher, C., [218](#)
Lustig, C. A., [401](#)
Lutzenberger, W., [158](#)
Lwill, F., [187](#)
Lynch, A., [483](#)
Lynch, G., [42](#), [64](#), [70](#)
Lyons, R., [125](#)–[126](#)

M

Ma, H. I., [344](#)
Macaulay, D., [435](#)
MacDonald, K., [302](#)
Machang'u, R. S., [187](#)
Mackintosh, N. J., [148](#), [149](#), [150](#), [153](#)
Macklin, M. L., [457](#), [460](#)
Madden, D. J., [536](#)
Maddison, S., [302](#)
Maddox, S. A., [526](#)
Magendie, F., [48](#)
Magill, R., [331](#)
Maguire, E. A., [44](#), [290](#), [314](#)
Mahr, J., [184](#)
Maier, S., [432](#)
Majewska, A., [528](#)
Majlessi, N., [532](#)
Major, A., [410](#)
Makris, N., [531](#)
Malcuit, G., [78](#)
Maldonado, H., [81](#)
Malenka, R. C., [540](#)
Maleszka, R., [526](#)
Malley, J. C., [461](#)
Malpass, R. S., [91](#)
Mandell, A. R., [519](#)
Mandic, M., [314](#)
Mandler, G., [88](#)
Mandolesi, L., [359](#)
Mangels, J. A., [404](#)
Mann, J., [333](#)
Manns, J., [312](#)
Mantell, J. T., [479](#)

Manuck, S. B., [524](#)
Maquet, P., [316](#)
Maramara, L. A., [401](#)
Marci, S., [55](#)
Marcus, E. A., [82](#), [100](#)
Maren, S., [442](#), [443](#), [458](#)
Maril, A., [306](#), [310](#), [406](#)
Marin, M. F., [458](#)
Marino, M., [349](#)
Markee, T., [523](#), [526](#)
Markowitsch, H., [320](#), [322](#), [391](#), [434](#), [435](#), [441](#)
Marler, P., [482](#), [510](#)
Marmar, C., [456](#)
Marquis, S., [321](#)
Marshall, B. S., [127](#)
Marshall, L., [68](#), [315](#), [317](#)
Marshall, V. J., [264](#), [267](#)
Marshall-Pescini, S., [472](#)
Marsland, S., [77](#)
Marteniuk, R. G., [344](#)
Martin, J. P., [319](#)
Martin, M., [285](#)
Martineau, J., [489](#), [495](#)
Martinek, T. J., [334](#)
Martínez-Cué, C., [541](#)
Martinussen, R., [410](#)
Martorella, E. A., [436](#)
Maruff, P., [541](#)
Masliah, E., [540](#)
Masse, N. Y., [364](#)
Massman, P., [394](#)
Mastaglia, F. L., [355](#)
Mateer, C. A., [493](#)
Mather, M., [518](#)
Matisse, H., [251](#)
Matson, J. L., [186](#)

Matsumoto, M., [213](#)
Mattes, R. D., [82](#)
Mattfeld, A. T., [55](#)
Matthes-von Cramon, G., [493](#)
Mattson, D. H., [168](#)
Matuz, T., [503](#)
Maugeais, R., [504](#)
Mauk, M. D., [158](#)
Maurer, D., [509](#)
May, A., [355](#)
May, F. S., [460](#)
Maynard, L., [411](#)
Mazzanti, C. M., [408](#)
McBride, W. J., [217](#)
McCabe, B. J., [43](#), [44](#)
McCabe, D. P., [519](#)
McCaffery, J. M., [524](#)
McClearn, G., [523](#), [526](#)
McClelland, J., [28](#), [64](#)
McCormick, D. A., [155](#), [156](#)
McCowan, B., [479](#)
McCullough, A. M., [449](#)
McCully, J., [167](#)
McCutcheon, L., [525](#)
McDaniel, M. A., [300](#)
McDonald, A., [408](#)
McDonald, B. C., [542](#), [543](#)
McDonald, R., [205](#), [351](#)
McDowd, J. M., [338](#)
McEwen, B., [450](#), [459](#), [532](#)
McGaugh, J. L., [55](#), [296](#), [313](#), [434](#), [435](#), [442](#), [447](#), [448–449](#), [452](#)
McGhee, D. E., [91](#)
McGonigal, A., [110](#)
McGregor, K. K., [512](#)
McGrew, W. C., [333](#)
McGue, M., [523](#)

McIntosh, A. M., [288](#)
McIntosh, J., [517](#)
McKay, H., [533](#)
McKeachie, W., [450](#)
McKee, A. C., [319](#)
McKelvie, P., [525](#)
McKenzie, S., [298](#)
McLaren, I. P. L., [241–242](#)
McLaren, R. P., [266](#)
McLaughlin, J., [532](#)
McLaughlin, K. A., [113](#), [460](#), [524](#)
McLaughlin, T., [542](#)
McMillan, B. D., [352](#)
McNall, C., [441](#)
McNally, R., [419](#)
McNaughton, B., [315](#)
McNaughton, B. L., [109](#), [535](#), [536](#)
McNeilly, A. S., [202](#)
McNelly, T. L., [345](#)
McNutt, A., [218](#)
McSweeney, F., [77](#)
Medina, L. D., [544](#)
Meeuwsen, H. J., [344](#)
Mehring, C., [345](#)
Meir Drexler, S., [451](#)
Meissner, C. A., [91](#)
Meltzoff, A. N., [479](#)
Memon, A., [295](#)
Mendes de Leon, C., [546](#)
Menon, V., [210](#), [514](#)
Mercado, E., III, [284](#), [333](#), [334](#), [479](#)
Mervis, C. B., [251](#)
Merzenich, M. M., [106](#), [263](#), [355](#), [356](#)
Metcalf, J., [519](#)
Metter, E. J., [320](#)
Metzger, L. J., [457](#), [460](#)

Meyer, E., [400](#)
Meyer, G., [357–358](#)
Meyer, M., [428](#)
Mez, J., [319](#)
Mézenge, F., [314](#)
Mfinanga, G., [187](#)
Mgode, G. F., [175](#), [187](#)
Micera, S., [364](#)
Michel, G. F., [509](#)
Mickel, S. F., [280](#), [338](#), [541](#)
Miezin, F. M., [405](#)
Milad, M. R., [458](#)
Miles, C., [532](#)
Millan, J. R., [364](#)
Millar, J., [320](#)
Miller, B., [523](#), [526](#)
Miller, D., [542](#)
Miller, E. K., [387](#), [391](#), [392](#), [397](#)
Miller, G., [26–28](#), [372](#), [373](#), [379](#)
Miller, J. P., [111](#)
Miller, K., [534](#)
Miller, N. E., [470](#)
Miller, P., [297](#)
Miller, R. R., [298](#)
Miller, T., [533](#)
Millikan, C. H., [320](#)
Milliken, G. W., [356](#)
Milner, B., [277](#), [278](#), [304](#), [383](#)
Miltner, W. H., [456](#)
Mineka, S., [455](#), [475](#)
Minozzi, S., [220](#)
Minshew, N. J., [494](#)
Miranda-Scippa, A., [456](#)
Misanin, J. R., [298](#)
Mischel, W., [214](#)
Mishkin, M., [307](#), [377–378](#), [517](#)

Mishra, J., [352](#)
Mitchell, C. J., [432](#)
Mitchell, J., [287](#)
Mitchelmore, C., [525](#)
Miya, D. Y., [109](#)
Miyatsu, T., [300](#)
Mize, J., [471](#)
Mizumori, S. J., [109](#)
Mobbs, D., [210](#)
Mobini, S., [201](#), [207](#)
Mobley, W. C., [539](#)
Modahl, C., [494](#)
Moir, R. D., [545](#)
Molaison, H. G., [277–278](#), [280](#), [305](#), [311–312](#), [322](#), [330](#), [338](#), [349](#)
Moldow, R. L., [532](#)
Molinari, M., [359](#)
Moncaster, J. A., [545](#)
Monet, C., [251](#)
Monfils, M. H., [444](#)
Moon, D. Y., [202](#)
Moon, J., [540](#)
Mooney, R., [491](#)
Moore, J., [161](#)
Moore, J. W., [133](#)
Moore, K. S., [401](#)
Moore, U., [147](#)
Moreno-Jiménez, E. P., [538](#)
Morey, R. A., [459](#)
Morgan, A., [346](#)
Morgan, C. L., [472](#), [479](#)
Morgan, O., [352](#)
Mori, E., [322](#)
Morishita, H., [104](#)
Morishita, W., [540](#)
Morris, C., [288](#)
Morris, J., [543](#)

Morris, R. G. M., [284](#), [298](#)
Morrison, J. H., [533](#), [535](#)
Morse, D., [516](#)
Mosconi, L., [543](#)
Moscovitch, M., [281–282](#), [288](#), [313](#), [314](#)
Mosier, K. M., [345](#)
Moss, C., [424](#)
Moss, M., [535](#)
Mottaghy, F. M., [489](#)
Mowrer, O. H., [431](#)
Mozart, W. A., [341](#)
Muenssinger, J., [503](#)
Mugikura, S., [322](#)
Muhlberger, A., [456](#)
Mulcare, S. P., [361](#)
Mulder, C., [175](#), [187](#)
Mullen, A., [290](#)
Muller, A., [521](#)
Muller, D., [451](#)
Müller, N. C. J., [290](#)
Muller, R. U., [109](#)
Muller, V., [333](#)
Mumford, J. A., [303–304](#)
Mur, M., [303](#)
Murdoch, M., [461](#)
Murphy, J. M., [217](#)
Murphy, K., [214](#)
Murray, A. D., [495](#)
Murray, B. D., [518](#)
Murray, S., [284](#), [333](#)
Musen, G., [280](#)
Mussa-Ivaldi, F. A., [345](#)
Mwimanzi, S., [175](#), [187](#)
Myers, C. E., [159](#), [265–266](#), [267–268](#), [270](#), [310](#), [354](#), [544](#)
Myers, N., [506](#)
Myerson, J., [201](#)

N

Naatanen, R., [106](#)
Nabavi, S., [444](#)
Nádasdy, Z., [315](#), [316](#)
Nadel, L., [288](#), [313](#), [314](#), [539](#)
Nader, K., [298](#)
Nagar, I., [297](#)
Nagell, K., [474](#)
Naghdi, N., [532](#)
Nagy, O., [268](#), [270](#)
Nair, H. P., [457](#)
Naj, A. C., [545](#)
Naqvi, N. H., [214](#), [218](#)
Narens, L., [299](#)
Nargeot, R., [210](#)
Naskar, S., [363](#)
National Down Syndrome Society, [539](#)
National Institutes of Health Consensus Conference, [297](#)
Nave, K. A., [42](#)
Nee, D. E., [401](#)
Neider, M. B., [351](#)
Neisser, U., [437](#)
Nelson, C. A., [524](#)
Nelson, K., [507](#)
Nelson, T. O., [299](#)
Nemanic, S., [109](#)
Neri, G., [539](#)
Neri, P., [359](#)
Nery-Fernandes, F., [456](#)
Netto, L. R., [458](#)
Netzer, W. J., [545](#)
Neufield, P., [296](#)
Neuringer, A., [250](#)

Newberg, W., [393](#)
Newell, A., [334](#)
Newport, E. L., [510](#), [512](#)
Newton, I., [10](#)
Nguyen, J., [540](#)
Nguyen, K., [300](#)
Nguyen, T., [110](#)
Nichol-Larsen, D. S., [111](#)
Nichols, S., [545](#)
Nicholson, D., [516](#)
Nickerson, R. S., [285](#)
Nicklaus, J., [517](#)
Nicolelis, M. A. L., [115](#), [364](#)
Nicoletti, F., [349](#)
Nielsen, S. E., [532](#)
Nielsen, T., [317](#)
Nikin, H., [395](#)
Nikolaev, A., [542](#)
Nikolich, K., [521](#)
Nishida, T., [333](#)
Nishitani, N., [495](#)
Nithianantharajah, J., [546](#)
Nitsch, R., [450](#)
Niv, Y., [459](#)
Noice, H., [345](#)
Noice, T., [345](#)
Nolen, T. G., [82](#), [100](#)
Noll, D. C., [408](#)
Nong, Y., [545](#)
Nordberg, C., [537](#)
Nordeen, E. J., [492](#)
Nordeen, K. W., [492](#)
Norman, D., [387](#)
Norrholm, S. D., [460](#)
North, N. C., [125–126](#)
Norton, L., [169](#)

Nottebohm, F., [537](#)

Nouvian, M., [202](#)

Nowakowski, R., [537](#)

Nowicki, S., [491](#)

Nowinski, C. J., [319](#)

Nudo, R. J., [356](#)

Nyberg, L., [404](#), [405](#)

O

Oakes, M. A., [292](#)
Obama, B., [295](#)
Oberauer, K., [379](#)
Oberbauer, A. M., [184](#)
Obeso, J. A., [361](#)
O'Brien, L., [531](#)
Ochsner, K., [306](#)
O'Connell, G., [266](#)
O'Doherty, D. C., [459](#)
O'Doherty, J. E., [364](#)
O'Doherty, J. P., [205](#), [206](#)
Ody, C., [403](#), [408](#)
Ohayon, M. M., [536](#)
Ohman, A., [83](#)
Ohno, H., [545](#)
Okaichi, H., [308](#)
O'Kane, G., [308](#)
O'Keefe, J., [107](#), [109](#)
Olds, J., [209](#)
O'Leary, D. D., [542](#)
Olendorf, R., [203](#)
Oleson, P. J., [411](#)
Olguin, R. S., [474](#)
Oliveira, I. R., [456](#)
Ollinger, J., [407](#), [408](#)
Olness, K., [168](#)
Olton, D., [308](#)
Omalu, B., [324](#)
Opitz, J. M., [539](#)
O'Regan, K., [502](#)
O'Reilly, J., [186](#)
Orr, S. P., [457](#), [458](#), [460](#)

Ortiz, M., [514](#), [531](#)

Otaka, S., [288](#)

Otten, L. J., [287](#)

Oullet, J., [322](#)

Over, R., [80](#)

Overman, W., [530](#)

Overmier, J., [432](#), [433](#)

Owen, A. M., [394](#), [399](#)

Owren, M. J., [428](#)

P

Pa, J., [544](#)
Pack, A., [284](#), [333](#)
Packard, M. G., [350](#)
Paczkowski, C., [505](#)
Pakkenberg, B., [531](#)
Pallas-Bazarra, N., [538](#)
Paller, K. A., [317](#)
Palmer, A., [62](#)
Palmer, L. C., [70](#)
Palminteri, S., [214](#)
Palmiter, R. D., [211](#)
Pang, P. T., [445](#)
Pankskepp, J., [428](#)
Pannu, J. K., [310](#)
Pantev, C., [355](#)
Papassotiropoulos, A., [521](#)
Paper, E., [184](#)
Papez, J., [439](#)
Paradis, C. M., [436](#)
Paré, D., [447](#), [449](#)
Pare-Blagnoev, E. J., [266](#)
Paredes, M. F., [537](#)
Park, K. J., [202](#)
Park, N., [434](#)
Park, S., [407](#)
Parker, A., [307](#)
Parker, D., [531](#)
Parker, L., [212](#)
Parkinson, J. A., [442](#)
Parks, R., [541](#)
Parlow, A. F., [202](#)
Pascual-Leone, A., [104](#)

Paton, J., [537](#)
Patterson, G., [210](#)
Patterson, M., [263](#)
Paul, M. J., [529](#), [537](#)
Pauli, P., [444](#), [456](#)
Paulsen, J. S., [338](#)
Paulson, G., [210](#)
Pavlicek, B., [214](#)
Pavlov, I., [6–7](#), [12](#), [17](#), [23](#), [36](#), [47](#), [48](#), [65–66](#), [96](#), [121–122](#), [124](#), [130](#), [133–134](#), [153](#), [177](#)
Paz, J., [540](#)
Pearce, A. J., [355](#)
Peck, C., [430](#)
Pedersen, N., [523](#), [526](#)
Pedreira, M., [81](#)
Peeters, F., [113](#)
Peigneux, P., [316](#)
Peisner-Feinberg, E., [44](#)
Pelletier, J., [447](#)
Pelletier, R., [336](#)
Penfield, W., [392–393](#)
Penhune, V. B., [331](#), [361](#)
Penn, S., [82](#)
Pennington, B. F., [494](#), [539](#)
Penrod, S. D., [295](#)
Pepperberg, I. M., [482](#)
Perdomo, C., [70](#)
Pereira, S., [317](#)
Perel, S., [364](#)
Perez, J., [494](#)
Peri, T., [457](#), [460](#)
Perlman, S. E., [457](#)
Perret, S. P., [158](#)
Perrett, D. I., [492–493](#), [494](#)
Perrin, F., [316](#)
Perris, E., [506](#)
Persico, A., [321](#)

Perucci, C., [220](#)
Pessiglione, M., [214](#)
Peters, A., [535](#)
Peters, L., [422](#)
Peters, S., [491](#)
Petersen, R. C., [320](#)
Petersen, S. E., [405](#)
Peterson, C., [434](#)
Peterson, D., [537](#)
Petok, J. R., [544](#)
Petrides, M., [382–383](#), [393–394](#), [399–400](#), [403](#), [405](#)
Petrie, M., [168](#)
Petrill, S., [523](#), [526](#)
Petrosini, L., [359](#)
Petscher, E. S., [190](#)
Pettigrew, T., [253](#)
Pfeifer, B., [392](#)
Pfordresher, P. Q., [479](#), [480](#)
Phan, K., [440](#)
Phelps, E. A., [436](#), [437](#), [448](#), [459](#)
Phillips, M., [446](#)
Phillips, R., [441](#), [442](#), [445](#)
Phillips, S., [532](#)
Piaget, J., [474](#)
Picard, R. W., [83](#)
Picasso, P., [251](#)
Pichiecchio, A., [321](#)
Pickrell, J., [294](#)
Pierce, L. J., [91](#)
Pietrzak, R. H., [461](#)
Piguet, O., [437](#)
Pilato, F., [321](#)
Pilz, K., [106](#)
Pine, D. S., [422](#)
Pineda, R., [545](#)
Pinsker, H., [98](#)

Pintar, J. E., [213](#)
Piolino, P., [517](#)
Pipitone, M. A., [359](#)
Pitman, R. K., [457](#), [458](#), [460](#)
Platel, H., [361](#)
Plato, [1](#), [2](#), [2f](#)
Plaud, J., [80](#)
Pleger, B., [106](#)
Plomin, R., [521](#), [526](#)
Ploog, B. O., [271](#)
Plumm, K. M., [185](#)
Plunkett, G. M., [483](#)
Plutchik, R., [406](#)
Poh, M. Z., [83](#)
Pohl, P. S., [338](#)
Poirier, V., [533](#)
Poldrack, R. A., [266](#), [303–304](#), [338](#), [353](#), [360](#), [406](#)
Polgár, P., [267](#), [268](#), [270](#)
Poling, A., [175](#), [186](#)
Pollak, P., [363](#)
Polusny, M. A., [461](#)
Pomerleau, A., [78](#)
Pomerleau, D., [516](#)
Pomplun, M., [341](#)
Poo, M., [521](#)
Poon, C-S., [82](#), [83](#), [101](#)
Port, R., [263](#)
Porter, C., [122](#)
Porter, D., [250](#)
Porter, J., [425](#)
Porter, S., [296](#)
Post, R., [113](#)
Postle, B. R., [380](#), [401](#)
Postman, L., [188](#)
Potenza, M. N., [216](#), [218](#), [221](#)
Potter, E., [543](#)

Potter, M. C., [537](#)
Poucet, B., [108](#)
Powell, C., [545](#)
Powell, R. A., [454](#)
Prabhakaran, V., [353](#), [406](#)
Prakash, R. S., [351](#)
Prasad, J., [346](#)
Prather, J. F., [491](#)
Pratt, R. D., [70](#)
Preissl, H., [503](#)
Pressman, S. D., [422](#)
Preston, K., [220](#)
Price, D. L., [517](#)
Price, J., [543](#)
Prince, S. E., [536](#)
Prins, B., [450](#)
Privat, A., [433](#)
Proteau, L., [344](#)
Proulx, C. D., [444](#)
Provence, S., [494](#)
Pryor, K., [186](#)
Przybyslawsky, J., [298](#)
Ptito, A., [393](#)
Puffenberger, S. S., [491](#)
Puryear, C. B., [445](#)
Putnam, A. L., [300](#)

Q

Qi, D., [537](#)

Quarantini, L. C., [458](#)

Quinette, P., [321](#)

Quirk, G., [447](#)

Quiroga, R., [302](#)

R

Rábano, A., [538](#)
Rabinovitch, M. S., [523](#)
Radak, Z., [545](#)
Radelet, M., [295](#)
Radin, A., [519](#)
Ragert, P., [106](#)
Raichle, M. F., [405](#)
Raine, A., [376](#)
Rakic, P., [526](#), [537](#)
Ramirez, A., [543](#)
Ramirez, S., [445](#)
Ramón y Cajal, S., [45](#)
Ramos, B. M. C., [169](#)
Ranganath, C., [88](#), [308](#), [314](#)
Ranganathan, R., [345](#)
Rankin, C. H., [77](#), [80](#), [82](#), [100](#)
Rao, A. K., [545](#)
Rapp, P., [533](#), [535](#)
Rasch, B. H., [317](#)
Rasch, M., [537](#)
Rasmussen, T., [535](#)
Rath, M. B., [461](#)
Ratner, H., [541](#)
Rauch, S. L., [267](#), [458](#)
Rauchs, G., [314](#)
Rauscher, F., [525](#)
Rawson, R., [220](#)
Ray, E., [245](#)
Raymaekers, R., [489](#)
Rayner, R., [12–13](#), [454](#)
Raz, N., [533](#)
Read, J., [294–295](#)

Reagan, R., [541](#)
Reale, M., [542](#)
Recanzone, G. H., [355](#)
Redd, W. H., [169](#)
Reddy, L., [302](#)
Redgrave, P., [362](#)
Redick, T. S., [352](#)
Redish, A. D., [109](#)
Reed, J., [312](#)
Reed, T., [523](#), [526](#)
Reeves, R., [540](#)
Regen, F., [542](#)
Reggers, J., [316](#)
Regis, J., [110](#)
Register-Mihalik, J. K., [319](#)
Reifsnider, E. S., [159](#)
Reingold, E. M., [341](#)
Reinhard, J., [202](#)
Reis, J., [68](#)
Reisberg, D., [434](#)
Reiss, A., [210](#), [514](#)
Reiss, D., [443](#), [479](#)
Remington, B., [167](#)
Renner, M., [44](#)
Rennie, C., [446](#)
Renoir, J., [251](#)
Rescorla, R., [138–140](#), [146](#), [148–149](#), [153](#), [170](#), [231–232](#)
Ressler, K. J., [460](#), [526](#)
Rethelyi, J., [268](#), [270](#)
Rex, C. S., [64](#)
Rey, C., [190](#)
Rey, V., [245](#)
Rey-Hipolito, C., [357–358](#)
Reynolds, V., [333](#)
Rhodes, M. G., [519](#)
Ribak, C. E., [537](#)

Ribot, T., [296](#), [311](#)
Richards, D. G., [479](#)
Richards, L. G., [338](#)
Richmond, M., [245](#)
Rideout, B., [525](#)
Rideout, H., [212](#)
Ridgway, S., [480](#)
Riehl, C., [203](#)
Riggio, L., [489](#)
Riley, D. O., [319](#)
Ringman, J. M., [544](#)
Risacher, S. L., [542](#), [543](#)
Rissman, J., [56](#)
Ritvo, E. R., [494](#)
Rizzolatti, G., [489](#), [490](#)
Robb, M. B., [525](#)
Robbins, T. W., [205](#), [216](#), [442](#)
Roberts, D., [477](#)
Roberts, J., [533](#)
Roberts, P., [167](#)
Roberts, W. A., [185](#), [283](#)
Robertson, E. M., [68](#), [306](#), [339](#)
Robinson, G., [308](#)
Robinson, S., [503](#)
Robinson, T. E., [113](#), [210](#), [211](#), [218](#), [427](#)
Robinson-Podolski, C., [344](#)
Roche, B., [431](#)
Rockland, C., [442](#), [445](#)
Rockstroh, B., [355](#)
Rodrigue, K. M., [533](#)
Rodriguez, M., [362](#), [538](#)
Rodriguez, M. L., [536](#)
Rodriguez-Oroz, M., [362](#)
Roediger, H. L., [289](#), [290](#), [295](#), [300](#)
Roesch, M. R., [206](#)
Roeyers, H., [489](#), [494](#)

Rogers, S. J., [494](#)
Roldan, E., [441](#)
Rolls, E. T., [302](#), [447](#)
Romanes, G. J., [479](#)
Romano, A., [81](#)
Roozendaal, B., [442](#), [450](#), [458](#)
Rosasco, M. L., [458](#)
Rosch, E., [251](#), [253](#)
Rosellini, R. A., [433](#)
Rosen, B. R., [215](#)
Rosen, J. B., [113](#)
Rosen, S., [210](#)
Rosenbaum, D. A., [330](#), [331](#)
Rosenbaum, P. S., [334](#)
Rosene, D., [535](#)
Rosenkranz, J., [443](#)
Rosenkranz, K., [356](#)
Rosenkranz, M., [451](#)
Rosenzweig, E. S., [109](#), [535](#)
Rosenzweig, M., [44](#)
Rosenzweig, R., [44](#)
Ross, D., [469](#)
Ross, S. A., [469](#)
Rosvold, H. E., [523](#)
Rotenberg, A., [109](#)
Roth, I., [243](#)
Rothbaum, B. O., [457](#), [459](#)
Rothwell, J. C., [68](#), [356](#)
Rotte, M., [306](#)
Rotter, J. B., [470](#)
Rouleau, I., [322](#)
Rounsaville, B., [218](#)
Routtenberg, A., [209](#)
Rovee-Collier, C., [505](#), [506](#)
Rozin, L., [166](#)
Rubin, D., [436–437](#)

Ruch, F., [517](#)
Rudge, P., [313](#)
Rudrauf, D., [218](#)
Rueda, N., [546](#)
Rugg, M. D., [287](#)
Ruiz, B. P., [158](#)
Rumelhart, D., [28–29](#), [30](#), [64](#), [149](#), [235](#)
Ruparel, K., [531](#)
Ruscio, A. M., [113](#)
Russell, A. F., [202](#)
Russon, A. E., [479](#)
Rusted, J., [541](#)
Rutland-Brown, W., [319](#)
Rutter, M., [524](#)
Ryan, L., [314](#), [435](#)
Ryan, P., [530](#)
Rymer, R., [510](#)

S

Sabb, F. W., [408](#)
Sadato, N., [104](#)
Saddiqui, S., [459](#)
Saenz, M., [104](#)
Sakamoto, A., [487](#)
Sakamoto, J., [250](#)
Salamone, J., [210](#), [211](#)
Salehi, A., [540](#)
Saletan, W., [295](#)
Salmon, D., [541](#), [545](#)
Salthouse, T. A., [516](#), [519](#)
Samsonovich, A., [314](#)
Sanders, G., [532](#)
Sandoval, K., [537](#)
Sandoval, M., [210](#), [211](#)
Sanes, J., [528](#)
Sanfey, A. G., [214](#)
Santangelo, G., [219](#)
Santangelo, V., [55](#)
Santorelli, S., [451](#)
Santori, A., [55](#)
Santucci, D. M., [364](#)
Sapolsky, R., [450](#), [451](#), [459](#)
Sara, S. J., [298](#)
Sarga, L., [545](#)
Sargent, J. D., [488](#)
Satterthwaite, T. D., [531](#)
Sattler, K. M. P., [189](#)
Saufley, W., [288](#)
Saunders, J. C., [432](#)
Save, E., [108](#)
Sawers, A., [337](#)

Saykin, A. J., [542](#), [543](#)
Sazma, M. A., [449](#)
Scahill, R., [313](#)
Scarmeas, N., [546](#)
Schachter, D. L., [322](#), [406](#)
Schachter, S., [422–423](#)
Schacter, D. L., [89](#), [280](#), [306](#), [310](#), [321](#), [404](#)
Schaechter, J. D., [112](#)
Schafe, G. E., [298](#)
Schaich-Borg, A., [213](#)
Schall, M., [531](#)
Schapiro, M., [545](#)
Schapiro, S. J., [475](#)
Schauer, F., [253](#)
Scheidt, R. A., [345](#)
Schellenberg, E. G., [525](#)
Schenker, N., [391](#)
Scherzer, P. B., [322](#)
Schiebel, A., [531](#)
Schiffer, R. B., [168](#)
Schinder, A. F., [537](#)
Schleger, F., [503](#)
Schliemann, T., [535](#)
Schlinger, H., [191](#)
Schlosser Covell, G. E., [545](#)
Schmid, S., [83](#), [540](#)
Schmidt, C., [540](#)
Schmidt, R. A., [334](#), [336](#), [337](#), [338](#)
Schmitz, C., [537](#)
Schmolck, H., [305](#), [312](#), [437](#)
Schneider, A., [521](#)
Schneider, J., [546](#)
Schneider, J. A., [515](#)
Schoenbaum, G., [206](#)
Schott, M., [175](#)
Schouten, D., [317](#)

Schreiner, T., [317](#)
Schry, A. R., [457](#)
Schuemie, M., [456](#)
Schuerbeeck, L. V., [494](#)
Schugens, M. M., [158](#)
Schuhmann, E., [530](#)
Schulkin, J., [113](#)
Schultz, J., [206](#)
Schultz, W., [207](#), [212](#)
Schumacher, J., [451](#)
Schutz, G., [165](#)
Schwabe, L., [450](#), [458](#)
Schwartz, A. B., [364](#)
Schwartz, B., [283](#)
Schwartz, J. L. K., [91](#)
Schwartz, M. D., [169](#)
Schwartzkroin, P., [532](#)
Schwarzenegger, A., [324](#)
Schweitzer, J. B., [184](#), [411](#)
Schwenkreis, P., [106](#)
Scoville, W., [277](#), [304](#)
Scribner, K., [203](#)
Scullin, M. K., [536](#)
Sears, L. L., [159](#)
Seger, C. A., [338](#), [353](#), [406](#)
Seidman, J., [346](#)
Seidman, L., [531](#)
Sekeres, M., [537](#)
Seligman, M. E., [432](#), [433](#), [434](#)
Sellitto, M., [208](#)
Semendeferi, K., [391](#)
Serena, J., [218](#)
Serrell, O., [35](#), [55](#)
Servatius, R. J., [532](#)
Seshadri, S., [545](#)
Seyfarth, R. M., [203](#)

Seymour, B., [203](#)
Shahan, T. A., [185](#), [186](#)
Shahidullah, S., [503](#)
Shalev, A., [456](#)
Shalev, A. Y., [457](#), [460](#)
Shallice, T., [387](#), [400](#)
Shanks, D. R., [89](#), [280](#)
Shannon, C., [27](#)
Shapiro, D. C., [335](#)
Sharot, T., [436](#), [448](#)
Shaw, G., [525](#)
Shaw, J., [296](#)
Shaw, L. M., [544](#)
Shaw, P., [403](#)
Sheffer, L., [299](#)
Sheffield, J., [533](#)
Shekhar, R., [320](#)
Shen, J., [535](#)
Shen, L., [542](#), [543](#)
Shenoy, K. V., [364](#)
Shenton, M., [459](#), [460](#)
Shepard, R., [230–231](#)
Sheppard, L., [541](#)
Sheridan, C. J., [503](#)
Sheridan, J., [451](#)
Sheridan, M. A., [460](#), [491](#), [524](#)
Sherman, G., [515](#)
Sherrington, C., [48](#), [51](#)
Sherwin, B., [532](#)
Sherwin, W. B., [333](#)
Shettleworth, S. J., [476](#)
Shibuya, A., [487](#)
Shields, G. S., [449](#)
Shiffrin, R., [370](#)
Shimamura, A. P., [393](#), [404](#)
Shin, L. M., [458](#)

Shin, R. K., [408](#)
Shirer, W. R., [290](#)
Shizgal, P., [219](#)
Shohamy, D., [147](#), [161](#), [265](#), [266](#), [269](#), [354](#), [544](#)
Shpaner, M., [215](#)
Shults, C., [541](#)
Sidman, R., [526](#)
Siegel, S., [166–168](#), [169](#)
Silva, A. J., [165](#)
Silva, Y., [218](#)
Silverman, I., [515](#)
Simeral, J. D., [364](#)
Simmons, R., [185](#)
Simões-Franklin, C., [215](#)
Simon, A. J., [542](#)
Simonet, P., [428](#)
Simons, D. J., [339](#)
Sinatra, F., [324](#)
Singer, J., [422–423](#)
Singer, R. A., [284](#)
Singh, T., [206](#)
Sinha, N., [291](#)
Sinigaglia, C., [490](#)
Sirota, A., [316](#)
Sistrunk, F., [183](#)
Skehel, P. A., [165](#)
Skerrett, D., [446](#)
Skilleter, A. J., [214](#)
Skinner, B. F., [13–14](#), [18](#), [24](#), [36](#), [126–127](#), [177](#), [178–180](#), [183](#), [186](#), [187](#), [200–201](#), [205](#), [219](#)
Slater, P., [297](#), [479](#)
Sloutsky, V. M., [507](#)
Slusarz, P., [338](#)
Small, S., [535](#)
Smith, A., [531](#)
Smith, D., [533](#)
Smith, E. E., [214](#), [249](#)

Smith, G., [542](#)
Smith, I. M., [494](#)
Smith, J. R., [185](#)
Smith, M., [459](#)
Smith, M. E., [304](#)
Smith, S., [288](#)
Smith, Y., [362](#)
Smithson, B., [454](#)
Smotherman, W., [503](#)
Smulofsky, J., [545](#)
Snaedal, J., [545](#)
Snape, M., [62](#)
Snow, C. E., [512](#), [513](#)
Snyder, A. Z., [407](#), [408](#)
Snyder, J. S., [537](#)
Soares, J. C., [494](#)
Sobczynska, M., [184](#)
Söderlund, H., [314](#)
Soderstrom, N. C., [285](#), [519](#)
Sohn, H., [202](#)
Sokolov, E. N., [96](#)
Solanto, M., [411](#)
Solomon, L. Z., [436](#)
Solomon, P., [161](#), [516](#)
Somaiya, R., [438](#)
Somogyi, E., [502](#)
Song, S. B., [339](#)
Sonuga-Barke, E. J. S., [524](#)
Sood, S. K., [363](#)
Sorensen, J., [535](#)
Sorrells, S. F., [537](#)
Soussignan, R., [422](#)
Southwick, S. M., [461](#)
Spalding, M. C., [364](#)
Speaker, C., [506](#)
Speekenbrink, M., [89](#)

Spence, K., [24](#), [477](#)
Spence, M., [503](#)
Spencer, W. A., [79](#), [80](#), [81](#), [96](#)
Sperling, G., [370–372](#)
Spiker, C., [245](#)
Spoelgen, R., [521](#)
Spring, J. D., [458](#)
Spritzer, M. D., [537](#)
Spurzheim, J., [40](#)
Squire, L. R., [88](#), [97](#), [100](#), [110](#), [280](#), [291](#), [292](#), [297](#), [305](#), [307](#), [312](#), [313](#), [320](#), [321](#), [322](#), [338](#), [354](#),
[393–394](#), [437](#)
Sripada, C. S., [457](#)
Stackman, R. W., Jr., [109](#)
Staddon, J., [188](#)
Stagner, J. P., [284](#)
Stak, C. E. L., [55](#)
Stalnaker, T. A., [206](#)
Stampe, D. M., [341](#)
Stanton, M., [505](#), [526](#)
Stanush, P. L., [345](#)
Stark, H. A., [89](#)
Stedron, J., [540](#)
Steele, C. J., [361](#)
Steele, J. W., [542](#)
Steele, K., [252](#), [525](#)
Steen, T. A., [434](#)
Stefanacci, L., [305](#), [312](#)
Stefanova, E., [521](#)
Stefanson, H., [545](#)
Steinberg, L., [529](#)
Steinberg, S., [256](#), [545](#)
Steinmetz, J. E., [154](#), [157](#)
Steinvorth, S., [314](#)
Stellman, S. D., [457](#)
Stelmach, G. E., [337](#)
Stenstrom, P., [317](#)

Stepper, S., [422](#)
Stern, R. A., [319](#)
Stern, S. A., [70](#), [546](#)
Stern, Y., [546](#)
Stevens, J. S., [459](#)
Steward, O., [308](#)
Stickgold, R., [346](#)
Stiers, W., [338](#)
Stoltz, S., [181](#)
Storie, D., [428](#)
Strack, F., [422](#)
Strange, B. A., [298](#)
Straub, R. E., [408](#)
Straube, T., [456](#)
Stravinsky, I., [250](#)
Stringer, T. P., [537](#)
Stroop, J. R., [387](#)
Strooper, B., [543](#)
Stroup, K., [433](#)
Stylopoulos, L., [542](#)
Subkov, A. A., [131](#)
Suddendorf, T., [494](#)
Sugiyama, Y., [333](#)
Suh, L. S., [536](#), [538](#)
Sullan, M. J., [319](#)
Sully, A., [175](#), [186](#)
Sun, R., [338](#)
Sunderland, A., [518](#)
Suñer-Soler, R., [218](#)
Sungkhasettee, V. W., [300](#)
Sur, M., [528](#)
Suris, A. M., [458](#)
Suster, J., [535](#)
Suthana, N., [55](#)
Sutherland, G. T., [536](#), [538](#)
Sutherland, R., [514](#), [531](#)

Sutton, M. A., [101](#)
Suzuki, M., [322](#)
Svirsky, M. A., [115](#)
Svoboda, K., [526](#)
Swaab, D., [532](#)
Swain, R. A., [359](#)
Swan, G., [523](#), [526](#)
Sweatt, J. D., [525](#)
Swede, H., [517](#)
Swendsen, J., [113](#)
Swenson, M., [338](#)
Swenson, N. C., [83](#)
Swerdlow, N. R., [83](#), [338](#)
Swing, E. L., [487](#)
Swinnen, S., [335](#)
Szabadi, E., [201](#), [207](#)

T

Taborsky, M., [203](#)
Taira, M., [54](#)
Tajudeen, B. A., [115](#)
Takagi, M., [322](#)
Takahashi, Y. K., [206](#)
Talarico, J., [436–437](#)
Tamres, L. K., [426](#)
Tan, H. Y., [408](#)
Tanaka, J. W., [91](#)
Tanaka, S. C., [206](#)
Tang, C., [447](#)
Tang, M., [546](#)
Tanimoto, E., [352](#)
Tannock, R., [411](#)
Tarr, M. J., [91](#)
Taskiran, D., [515](#)
Taub, E., [111](#), [112](#), [355](#)
Taylor, J. R., [298](#)
Taylor, L., [446–447](#)
Taylor, S., [440](#)
Taylor, S. E., [426](#)
Teasdale, J., [435](#)
Technau, G., [44](#)
Tees, R. C., [511](#)
Tegenthoff, M., [106](#)
Teixidor, P., [472–473](#)
Telang, F., [411](#)
Telemann, G., [250](#)
Tendolkar, I., [298](#)
Tennant, F., [220](#)
Terrace, H., [242–243](#)
Terreros-Roncal, J., [538](#)

Terry, C., [338](#)
Tesch-Romer, C., [340](#)
Tessier-Lavigne, M., [542](#)
Teter, B., [545](#)
Teuber, J., [278](#)
Thatcher, R., [393](#)
Thickbroom, G. W., [355](#)
Thiery, E., [113](#)
Thiruchselvam, R., [446](#)
Thomas, A. K., [295](#)
Thomas, M. S. C., [509](#), [512](#)
Thompson, C., [437](#)
Thompson, D., [287](#), [295](#)
Thompson, E. F., [115](#)
Thompson, P. A., [111](#)
Thompson, P. M., [543](#)
Thompson, R. F., [77](#), [79](#), [80](#), [81](#), [84–85](#), [96](#), [153](#), [155](#), [156](#), [157](#), [158](#), [159–160](#), [244](#), [260–261](#),
[516](#), [532](#), [533](#)
Thompson, S. A., [314](#)
Thompson, T., [436](#)
Thompson, W. F., [525](#)
Thompson-Schill, S. L., [405](#)
Thomson, H., [290](#)
Thomson, N., [376](#)
Thorndike, E. L., [6](#), [8](#), [176–177](#), [178–179](#), [182](#), [187](#), [188](#), [235](#), [334](#), [344](#), [468](#), [470](#), [472](#), [479](#),
[480–481](#)
Thorpe, S. J., [302](#)
Thorpe, W. H., [472](#), [477](#), [482](#)
Thuras, P., [461](#)
Tibbo-Valeriotte, H. L., [319](#)
Tigges, J., [535](#)
Tillein, J., [115](#)
Timberlake, W., [199](#)
Tinbergen, N., [94–95](#)
Titone, D., [269](#)
Titz, C., [518](#)

Todd, M., [480](#)
Todd, R., [446](#)
Tolman, E., [17–19](#), [93](#), [177](#)
Tomasello, M., [474](#)
Tompary, A., [306](#)
Tomsic, D., [81](#)
Tonegawa, S., [445](#)
Toni, N., [537](#)
Tononi, G., [315](#)
Tops, M., [317](#)
Torrealba, F., [218](#)
Trabasso, T. R., [143–144](#)
Trachtenberg, J., [528](#)
Tranel, D., [218](#), [338](#), [442](#), [445](#)
Trauner, D., [545](#)
Travers, J., [210](#)
Travers, S., [210](#)
Treffert, G., [35](#)
Trejo, J. L., [546](#)
Tremblay, L., [207](#)
Trezniak, C., [456](#)
Trivers, R., [203](#)
Trojano, L., [219](#)
Trojanowski, J. Q., [544](#)
Trombly, C. A., [344](#)
Tronson, N. C., [298](#)
Tryon, R., [521](#)
Tsai, W., [546](#)
Tully, T., [523](#)
Tulving, E., [89](#), [278](#), [279](#), [282–283](#), [286](#), [287](#), [322](#), [404](#), [405](#)
Tune, L. E., [411](#)
Turner, A. P., [334](#)
Turner, J., [435](#)
Turner, R. S., [349](#), [357–358](#)
Tuszynski, M., [533](#)
Tybulewicz, V. L. J., [539](#)

Tyler, A. N., [509](#)

Tyler-Kabara, E. C., [364](#)

U

Ulfhake, B., [517](#)

Umbricht, A., [220](#)

Uncapher, M., [435](#)

Underwood, M., [441](#)

Ungerleider, L. G., [331](#), [357–358](#)

UNICEF, [189](#)

Unsworth, N., [352](#)

Urs, R., [543](#)

Urschel, J., [331](#)

Uswatte, G., [111](#)

Uyeyama, R., [284](#), [333](#)

V

Vaegan, T., [509](#)
Vakil, E., [297](#)
Valdizán, E., [546](#)
Valentine, E., [290](#)
Vallina, I., [540](#)
van Buskirk, R., [449](#)
van de Beck, C., [530](#)
VandenBerg, P. M., [358](#)
Van de Poll, N., [532](#)
van der Mast, C., [456](#)
van Goozen, S., [532](#)
van Lehn, K., [330](#)
van Os, J., [113](#)
van Praag, H., [537](#)
van Rooij, S. J. H., [459](#)
van Rossum, J. H. A., [337](#)
Vanvuchelen, M., [494](#)
van Waarde, J. A., [298](#)
van Wingen, G. A., [298](#)
Varner, J., [210](#)
Vecchi, S., [220](#)
Vehrencamp, S. L., [482](#)
Veit, R., [106](#)
Velliste, M., [364](#)
Verhaeghen, P., [518](#)
Verhagen, R., [187](#)
Verma, R., [531](#)
Versteeg, D., [428](#)
Verster, A., [220](#)
Viader, F., [321](#)
Vicari, S., [539](#)
Vieth, W., [476](#)

Vilcinskas, A., [175](#)
Villado, A. J., [336](#)
Villar, A., [540](#)
Villeda, S. A., [535](#)
Vindras, P., [349](#)
Viskontas, I. V., [314](#)
Vitale, C., [219](#)
Vitiello, M. V., [536](#)
Vivar, C., [537](#)
Vogel, E. K., [379](#)
Vogt, B. A., [215](#)
Volkow, N., [411](#)
Voll, C. A., Jr., [344](#)
Vollono, C., [321](#)
von Cramon, D. Y., [493](#)
von Oertzen, T., [334](#)
Voss, J. L., [317](#)
Voss, M. W., [351](#)
Vossel, G., [437](#)
Votaw, J. R., [349](#)

W

Wacker-Gussman, A., [503](#)

Wade, K., [294–295](#)

Wager, T. D., [214](#)

Wagner, A., [138–140](#), [146](#), [148–149](#), [153](#), [170](#), [231–232](#), [269](#), [287](#), [306](#), [310](#)

Wagner, A. D., [304](#), [404](#)

Wagner, A. R., [406](#)

Wagner, H. R., [457](#)

Wagner, I. C., [290](#)

Wagner, T., [440](#)

Wahaba, W. W., [166](#)

Waiter, G. D., [495](#)

Wake, H., [42](#)

Wakita, M., [250](#)

Walczak, M., [184](#)

Wald, M. M., [319](#)

Waldron, E., [205](#)

Walicke, P., [541](#)

Walk, R., [89](#), [91](#)

Walker, D. M., [529](#)

Walker, L. C., [517](#), [542](#)

Walker, M. P., [346](#)

Wall, A. M., [168](#)

Wallace, D. S., [422](#)

Wallis, J. D., [387](#), [391](#), [392](#)

Walls, R., [183](#)

Walsh, V., [490](#)

Waltz, J. A., [267](#)

Waltzman, S. B., [115](#)

Wang, G., [411](#)

Wang, L. S., [545](#)

Wang, P., [322](#)

Wang, S., [537](#)

Wang, S.-H., [284](#), [298](#)
Wang, W., [364](#)
Ward, K. E., [109](#)
Ward-Robinson, J., [264](#), [267](#)
Waring, J. D., [437](#)
Warner, E. E., [200](#)
Washington, D., [324](#)
Washington, G., [282](#)
Watanabe, S., [250](#)
Waterhouse, L., [494](#)
Watson, J., [11–13](#), [36–37](#), [454](#)
Watts, K., [518](#)
Wearing, C., [21](#), [22](#), [35](#)
Wearing, D., [22](#)
Weatherly, J. N., [185](#)
Weber, D. J., [364](#)
Weber, M., [450](#)
Weeks, D. L., [334](#)
Weetjens, B. J., [175](#), [186](#), [187](#)
Weidemann, G., [432](#)
Weiland, N., [532](#)
Weinberger, D., [521](#)
Weinberger, D. R., [407](#), [408](#), [409](#)
Weinberger, N. M., [106](#), [261–262](#), [263](#), [442](#)
Weiner, M. W., [544](#)
Weinstein, A., [216](#), [221](#)
Welker, E., [528](#)
Wellik, K. E., [545](#)
Wells, G. L., [295](#)
Wells, S., [533](#)
Wendling, F., [110](#)
Werchan, D. M., [506](#)
Werker, J. F., [511](#)
Werner-Seidler, A., [458](#)
Wernert, L., [525](#)
West, J. D., [542](#), [543](#)

West, K., [82](#)
West, M., [535](#)
West, R., [44](#)
West, S. A., [202](#)
Westbrook, R., [458](#)
Westerberg, H., [411](#)
Wharton, D. M., [544](#)
Wheatley, E. G., [535](#)
Whiskin, E. E., [485](#)
Whitaker, C., [460](#)
White, N. M., [205](#), [350](#), [351](#)
Whitehead, A., [70](#)
Whitehorn, D., [441](#)
Whiten, A., [333](#), [472–473](#), [475](#), [494](#), [495](#)
Whitford, A., [364](#)
Wichers, M., [113](#)
Wichmann, T., [362](#)
Wickelgren, W., [293](#)
Wiedeman, G., [444](#), [456](#)
Wienbruch, C., [355](#)
Wiersema, J. R., [489](#)
Wiesel, T., [509](#), [528](#)
Wieser, J., [345](#)
Wightman, D., [183](#)
Wigmore, S. W., [484](#)
Wilcock, G. K., [62](#), [70](#)
Wilcoxon, L., [455](#)
Wildgruber, D., [428](#)
Wilding, J., [290](#)
Wilkes, F., [525](#), [526](#)
Wilkinson, D., [542](#)
Will, B., [308](#)
Willeumier, K., [393](#)
Williamon, A., [356](#)
Williams, A., [494](#)
Williams, A. M., [341](#)

Williams, B., [438](#)
Williams, B. A., [432](#)
Williams, D. L., [494](#)
Williams, J. H., [341](#), [495](#)
Williams, K. D., [214](#)
Williams, L., [446](#)
Williams, P., [393](#)
Williamson, A., [533](#)
Willing, J., [529](#), [537](#)
Willingham, D. B., [338](#)
Wills, A. J., [241–242](#), [266](#)
Wills, T., [109](#)
Wilson, B. A., [22](#), [243](#)
Wilson, C., [302](#)
Wilson, D. A., [77](#)
Wilson, M., [315](#)
Wilson, M. A., [315](#), [316](#), [317](#)
Wilson, R., [546](#)
Wilson, R. S., [515](#)
Wilson, T., [525](#)
Winek, C. L., [166](#)
Winocur, G., [281–282](#), [288](#), [313](#), [314](#), [537](#)
Winstein, C. J., [111](#)
Winter, W., [524](#)
Wiseman, F. K., [539](#)
Wishart, J. G., [243](#)
Wiskott, L., [537](#)
Wittenberg, G. F., [112](#)
Witthoft, N., [304](#)
Wittlinger, R., [289](#)
Wixted, J. T., [110](#)
Wojtowicz, J. M., [537](#)
Wolf, O. T., [451](#)
Wolf, P., [523](#), [526](#)
Wolf, S. L., [111](#)
Wollmer, M., [521](#)

Wolpert, D. W., [345](#)
Wolz, J. P., [479](#)
Wong, L., [545](#)
Wood, D., [474](#)
Wood, N. E., [458](#)
Woodlinger, B., [364](#)
Woodruff, B. K., [545](#)
Woodruff-Pak, D. S., [158](#), [516](#), [533](#)
Woodworth, R., [344](#)
Woolley, C., [532](#)
Wooten, J. D., [319](#)
Wright, D. B., [113](#)
Wrisberg, C. A., [334](#)
Wu, C-F., [77](#)
Wu, J., [393](#)
Wulf, G., [335](#), [337](#), [338](#)

X

Xie, X., [532](#)

Xu, F., [506](#)

Xu, J., [532](#)

Xue, G., [303](#)–[304](#)

Y

Yadegarfar, G., [542](#)

Yanagihara, T., [320](#)

Yang, Y., [321](#)

Yarkoni, T., [214](#)

Yarrow, K., [339](#)

Yi, H., [507](#)

Yilmaz, O., [515](#)

Yin, H. H., [36](#)

Yonelinas, A. P., [88](#), [308](#), [314](#), [449](#)

Yoshida, W., [203](#)

Youn, G., [80](#)

Young, A., [314](#)

Young, A. J., [202](#)

Young, D. E., [335](#)

Young, D. L., [82](#), [83](#), [101](#)

Young, L. J., [427](#)

Yu, K., [283–284](#)

Yu, Q., [517](#)

Z

Zajonc, R., [422](#)

Zane, T., [183](#)

Zbrozyna, A., [447](#)

Zeanah, C. H., [524](#)

Zec, R. F., [407](#)

Zentall, T. R., [284](#), [473](#)

Zetterberg, H., [319](#), [545](#)

Zhao, C., [537](#)

Zhao, Z., [306](#)

Zhou, W., [284](#)

Zilov, G. N., [131](#)

Zimmer, J., [535](#)

Zimmerman, E., [428](#)

Zouzounis, J. A., [297](#), [320](#), [321](#)

Zubek, J., [523](#)

Zuniga, E., [540](#)

Zwaan, R. A., [422](#)

SUBJECT INDEX

A

Acetylcholine (ACh), [262](#)

Acoustic startle reflex, [77](#), [78f](#)

Acquired equivalence, [245–246](#), [267–269](#), [505–506](#)

Acquired savant syndrome, [35](#)

Action potential, [41](#)

Activity-dependent enhancement, [162–163](#)

Addiction

behavioral, [218–219](#)

classical conditioning in tolerance to drugs, [166–168](#)

pathological, [216–218](#)

ADHD. *See* [Attention-deficit/hyperactivity disorder \(ADHD\)](#)

Adolescence/adolescents, [512–515](#)

brain in, [529–533](#)

defined, [513](#)

male–female differences in learning and memory, [514–515](#)

maturation of working memory, [513–514](#)

prefrontal cortex in, [529–530](#)

sex hormones, effects on brain organization, [530–532](#)

Adulthood/adults

adolescence in crossing from childhood to, [512–515](#)

aging memory through, [515–519](#)

brain in, [533–538](#)

conditioning in aging, [516–517](#)

metamemory in, [518](#)

neurogenesis in, [536–538](#)

skill learning in aging, [517](#)

Adult neurogenesis, [536–538](#)

Advertising, behavior influenced by, [488](#)

African pouched rats, [175](#), [176](#), [176f](#), [184](#)

Aging and metamemory, [518–519](#). *See also* [Aging memory](#); [Development and aging](#)

Aging memory. *See also* [Development and aging](#)

from adulthood through old age, [515–519](#)

- conditioning in aging adults, [516–517](#)
- episodic and semantic memory, [517–518](#)
- localized neuron and synapse loss, [533–535](#), [534f](#)
- loss of synaptic stability, [535–536](#)
- metamemory and aging, [518–519](#)
- old memories vs. new learning and, [517–518](#)
- skill learning in aging adults, [517](#)
- working memory in aging adults, [516–517](#)

Agoraphobia, [453](#), [454](#)

Allele, [521](#)

Altruism, [202–204](#)

Alzheimer's disease, [541–547](#)

- defined, [541](#)
- diagnosis of, [543–544](#)
- Down syndrome and, [545–546](#)
- genetic basis of, [544–545](#)
- memory loss and cognitive deterioration in, [541–542](#)
- mental exercise to protect against, [546](#)
- plaques and tangles in brain, [542–544](#)
- treatment of, [547](#)

Amazon.com, [234](#)

American Sign Language (ASL), [502–503](#), [503f](#)

Amnesia, [277–278](#)

- anterograde, [305](#)
- functional, [321–323](#)
- as plot device, [323–324](#)
- retrograde, [311–312](#)
- transient global, [319](#), [320–321](#)

Amygdala, [39](#), [39f](#), [439f](#), [440f](#)

- anatomy of, [440–441](#)
- emotional influences on learning and memory, [439](#)
- emotional learning pathways in, [442–443](#)
- episodic memory storage and, [445](#)
- in expression of emotional responses, [441–442](#)
- retrieval and reconsolidation of memories and, [450–452](#)
- stress hormones and, [448–451](#)

Amyloid plaque, [542–544](#), [542f](#)

Androgens, [515](#)

Animals

cognitive skill memory in, [333–334](#)

emotional regulation in, [452](#)

emotions in, [424–428](#)

episodic memory in, [282–284](#), [307–308](#)

frontal lobes, comparative anatomy of, [392f](#)

hippocampus in, [106–107](#), [107f](#), [160f](#)

models of Down syndrome, [540–541](#)

robotic limbs for, [364](#)

semantic memory in, [282](#), [307–308](#)

social learning and memory in, [467](#)

song learning in, [481–483](#)

vocalizing without imitating, [480–481](#)

Anterior communicating artery (ACoA), [310](#)

Anterograde amnesia, [305](#)

Antidepressants, [69](#)

Anxiety, sensitization to, [109–110](#)

Apathy, [36](#)

Aplysia

classical conditioning in, [117](#), [162–163](#), [162t](#)

conditioned emotional responses in, [429–430](#)

habituation in, [98–99](#), [162t](#)

sensitization in, [99–100](#), [100t](#)

varieties of learning in, [162t](#)

Apoptosis, [527](#)

Appetitive conditioning, [125–126](#), [130](#), [130t](#)

Aristotle, [1–2](#), [2f](#), [3](#), [10](#), [31t](#)

Arousal, [420](#)

autonomic, [420–421](#)

Association, [1–3](#), [135](#)

extinguishing old, [133–134](#)

learned, [179–181](#)

Association cortex, [301–302](#)

Associationism, [1–2](#), [6–8](#)

Associative bias, [151–152](#)
Associative stage, [340](#)
Associative weight, [140](#)
Attention-deficit/hyperactivity disorder (ADHD), [410–412](#)
Autism spectrum disorder (autism)
 generalization and, [270–272](#)
 social learning and, [494–495](#)
Autonomic arousal, [420–421](#)
Autonomous stage, [340](#)
Aversive conditioning, [126–127](#), [130](#)
Avoidance conditioning, [430–432](#), [431f](#)
Avoidance learning, theories of, [431–432](#), [431f](#)
Axon, [41](#), [42f](#)

B

Backward chaining, [183](#)

Baddeley's model of working memory, [374–375](#)

Bandura's Bobo doll experiment, [469–470](#), [486](#)

Basal forebrain, [310–311](#)

Basal ganglia, [39](#), [39f](#), [205](#)

- brain activity during cognitive skill learning, [353–354](#)

- defined, [348](#)

- learning deficits after lesions, [349–351](#)

- neural activity during perceptual-motor skill learning, [351–353](#)

- and skill learning, [348–354](#)

Basolateral amygdala, [441](#), [443–444](#)

Behavior

- behavioral economics and, [199–201](#), [200f](#)

- changes following frontal lobe damage, [392–393](#)

- choice, [198–204](#)

- differential reinforcement of alternative, [189–190](#)

- media's impact on, [486–488](#), [487f](#)

- social manipulation of, [486–487](#)

- stimulation as substitute for training, [157–158](#)

- variable-interval schedules and matching law, [198–199](#), [198f](#)

Behavioral addiction, [218–219](#)

Behavioral economics, [199–201](#), [200f](#)

Behavioral processes

- classical conditioning and, [123–153](#)

- cognitive control, [370–391](#)

- concept formation, [226–275](#)

- development and aging and, [502–520](#)

- discrimination learning, [226–275](#)

- emotional influences on learning and memory, [418–439](#)

- episodic and semantic memory, [276–327](#)

- familiarization, [75–119](#)

- generalization, [226–275](#)

- habituation, [75–119](#)
- operant conditioning and, [176–204](#)
- semantic memory and, [276–327](#)
- skill memory, [330–347](#)
- social learning and memory, [468–488](#)
- working memory, [370–391](#)

Behaviorism, [11–14](#)

- cognitive approach to, [30](#)
- defined, [11](#)
- Estes and, [24–26](#)
- Hull and, [23–24](#)
- mathematical models of learning and, [23–24](#)
- neo-, [17–19](#)
- radical, [13–14](#)
- Skinner and, [13–14](#)
- Tolman and, [17–19](#)
- Watson and, [11–13](#), [36–37](#), [454](#)

Bell–Magendie law of neural specialization, [48](#)

Bias

- associative, [151–152](#)
- confirmation, [253](#)
- unconscious racial, [91](#)

Biochemical control of brain states, [68–70](#)

Blank slate, [10–11](#)

Blind design, [21](#)

Bliss point, [199–201](#)

Blocking effect

- classical conditioning and, [136–138](#), [142](#)
- contiguity and, [138](#)
- defined, [137](#)
- Kamin’s, [136–138](#), [137t](#)

Blocking experiment, [137–138](#), [140](#), [141–142](#)

Brain, [37–46](#)

- activity during cognitive skill learning, [353–354](#)
- adult neurogenesis and, [536–538](#)
- biochemical control of states in, [68–70](#)

- comparative neuroanatomy of, [38–42](#), [38f](#)
- concussion, [319](#)
- deep stimulation of, [363](#), [363f](#)
- Down syndrome, abnormalities and memory impairments in, [539–540](#)
- experience in changing structure of, [43–46](#)
- functions of, [47–57](#)
- human, [38–39](#), [39f](#)
- imaging, [39–41](#), [41f](#)
- injury effects on, [35](#), [58](#), [59](#)
- lesions of, [349–351](#)
- neurons and synapses in developing, [526–528](#)
- neurons in, [41–42](#), [42f](#)
- old, [536–538](#)
- plaques and tangles in, [542–544](#)
- plasticity, [43](#)
- punishment signaling in, [213–215](#)
- regions, [70–71](#), [71f](#)
- remembering and activity in, [55–57](#), [56f](#)
- song learning in birds and, [491–492](#), [491f](#)
- stimulation as substitute for behavioral training, [157–158](#)
- video games and, [352](#)
- “wanting” and “liking” in, [209–213](#), [209f](#), [211f](#), [216–219](#)

Brain-derived neurotrophic factor (BDNF), [520–521](#)

Brain from adulthood to old age, [533–538](#)

- localized neuron and synapse loss, [533–535](#), [534f](#)
- loss of synaptic stability, [535–536](#)
- neurogenesis and, [536–538](#)

Brain functions, [47–57](#)

- biochemical control of brain states, [68–70](#)
- brain injury effects, [35](#), [58](#), [59](#)
- electromagnetic stimulation of neurons, [65–68](#)
- incoming stimuli, [49](#), [49f](#)
- motor control, [49–51](#), [50f](#)
- neuron connections, [45–46](#), [45f](#)
- outgoing responses, [49–51](#), [50f](#)
- pathways into brain, [49](#), [49f](#)

in remembering, [55–57](#), [56f](#)

research, [65–70](#)

synapse, [60–62](#), [61f](#)

Brain in adolescence, [529–533](#)

in prefrontal cortex, [529–530](#)

sex hormones on brain organization, [530–532](#)

Brain substrates

classical conditioning and, [153–165](#)

cognitive control, [391–407](#)

concept formation, [255–266](#)

development and aging, [520–538](#)

discrimination learning, [255–266](#)

emotional influences on learning and memory, [439–453](#)

episodic and semantic memory, [301–318](#)

familiarization, [96–110](#)

generalization, [255–266](#)

habituation, [96–110](#)

operant conditioning and, [205–215](#)

semantic memory, [301–304](#)

skill memory and, [348–362](#)

social learning and memory, [489–492](#)

working memory, [391–407](#)

Brainstem, [39](#)

C

Cannon–Bard theory of emotion, [421f](#), [422](#)

Categorization task, [145f](#)

Category/categorization, [249](#)

Category learning, [249–252](#)

 in categorization, [249–252](#)

 defined, [249](#)

 network model of, [145f](#)

 prototypes and structure of natural categories, [251–252](#)

 stereotypes, discrimination, racism, and, [252–255](#)

 through discrimination learning, [250–251](#)

Caudate nucleus, [205](#)

Causality, [144–145](#)

Cell body, [41](#), [42f](#)

Central executive, [375](#)

Central nervous system (CNS), [37](#), [37f](#)

Central nucleus, [440–441](#)

Cerebellum, [39](#), [153–154](#)

 classical conditioning and, [39](#), [71](#)

 electrophysiological recordings in, [155–156](#), [156f](#)

 impaired conditioning following damage to, [158](#)

 and timing, [358–362](#)

Cerebral cortex, [38](#)

 searching for memories in, [58–60](#)

 and storage of skill memory, [356–358](#), [357f](#)

Chaining, [183](#)

Choice behavior, [198–204](#)

 behavioral economics and, [199–201](#), [200f](#)

 variable-interval schedules and matching law, [198–199f](#)

Chromosomes, [520](#). *See also* [Genetics](#)

Chronic traumatic encephalopathy (CTE), [319](#)

Classical conditioning, [6–7](#), [12](#), [17](#), [65–66](#), [120–173](#). *See also* [Conditioning](#); [Pavlovian conditioning](#)

Aplysia and, [117](#), [162–163](#), [162t](#)
appetitive, [130](#), [130t](#)
associative bias and ecological constraints, [151–152](#)
aversive, [130](#), [130t](#)
avoidance learning and, theories of, [431–432](#), [431f](#)
behavioral processes and, [123–153](#)
blocking effect and, [136–138](#), [142](#)
brain lesions and, [349–350](#)
brain substrates and, [153–165](#)
cerebellum and, [39](#), [71](#)
clinical perspectives on, [166–168](#)
conditioned emotional responses and, [428–434](#), [488](#)
defined, [122](#)
determinants of, [149–152](#)
drug addiction, in tolerance to, [166–168](#)
drug addiction and, [216–218](#), [217f](#)
electrophysiological recordings in cerebellum during, [155–156](#), [156f](#)
error correction and modulation of unconditioned stimulus, [135–146](#)
in everyday life, [130](#)
extinction and, [177–180](#)
eyeblick conditioning and, [127–131](#), [178](#)
hippocampus and, [160–161](#), [263–265](#), [304](#), [449](#)
imprinting and, [43](#)
in infants, [505](#)
invertebrates and cellular basis of learning, [161–165](#)
learned helplessness and, [431–432](#), [431f](#)
Mackintosh model of, [149](#)
mammalian conditioning of motor reflexes, [153–161](#)
medication reduction through, [168–169](#)
negative patterning and, [246–247](#)
observational conditioning and, [476](#)
operant conditioning and, [8](#), [175–176](#), [495](#)
overshadowing in, [134–135](#)
Pavlov's principle of, [122](#), [130t](#), [176–177](#)
phobias and, [454–456](#)
posttraumatic stress disorder and, [457](#)

Rescorla–Wagner model of, [138](#), [143](#), [149](#), [231–232](#)
skin conductance response and, [441–442](#), [442f](#)
stimulus attention and modulation of conditioned stimulus, [146–149](#)
temporal contiguity in, [190–191](#)
terminology of, [124t](#)
timing and, [150–151](#)
widely used procedures for, [130t](#)

Classical conditioning concepts, [123–131](#)

appetitive conditioning, [125–126](#)
aversive conditioning, [126–127](#)
conditioned response, [123–125](#)
conditioned stimulus, [123–125](#)
dog salivation, [123–125](#)
eyeblick conditioning, [127–131](#)
learning new association, [130–131](#)
mammalian conditioning of motor reflexes, [127–131](#)
unconditioned stimulus, [123–125](#)

Classical conditioning principles, [131–135](#)

compound conditioning, [134–135](#)
conditioned compensatory response, [131–132](#)
cues for conditioned stimulus, [132–133](#)
cues for unconditioned stimulus, [132–133](#)
extinguishing old association, [133–134](#)
overshadowing, [134–135](#)

Clinical perspectives

on classical conditioning, [166–168](#)
on cognitive control, [407–412](#)
on concept formation, [266–273](#)
on development and aging, [538–547](#)
on discrimination learning, [266–273](#)
on emotional influences on learning and memory, [453–461](#)
on episodic and semantic memory, [319–323](#)
on generalization, [266–273](#)
on habituation, [110–116](#)
on operant conditioning, [216–221](#)
on semantic memory, [319–323](#)

- on skill memory, [362–365](#)
- on social learning and memory, [492–495](#)
- on working memory, [407–412](#)

Closed skills, [331](#), [332](#)

Cochlear implant, [114–115](#), [114f](#)

Cognitive approach, [30](#)

- in addiction treatment, [219–221](#)
- to behaviorism, [30](#)
- mathematical psychology and, [24–26](#)

Cognitive control, [381–391](#)

- attention deficit/hyperactivity disorder, [410–412](#)
- behavioral processes and, [370–391](#)
- brain substrates and, [391–407](#)
- clinical perspectives on, [407–412](#)
- controlled updating of short-term memory buffers, [381–384](#)
- defined, [370](#)
- frontal brain activity during working-memory tasks, [395–398](#)
- frontal lobes and consequences of frontal-lobe damage, [391–395](#)
- intelligence, working memory, and, [388–389](#)
- mapping onto prefrontal cortex, [398–403](#)
- prefrontal control of long-term declarative memory, [403–407](#)
- prefrontal cortex in schizophrenia, [407–409](#)
- setting and planning goals, [384–385](#)
- stimulus selection and response inhibition, [386–388](#)
- task switching, [385–386](#)
- transient memories, [370–374](#)
- working memory, [374–380](#)

Cognitive deterioration, [541–543](#)

Cognitive expectancies, [432](#)

Cognitive map, [17–19](#), [19f](#), [109](#)

Cognitive psychology, [27](#), [145](#), [149](#)

Cognitive skills, [330](#)

- cerebellar activation during learning, [359–361](#), [361f](#)
- gender differences and, [343](#)
- learning, [353–354](#)

Cognitive stage, [340](#)

Collaborative filtering, [234](#)

Compensatory response, conditioned, [131–132](#)

Complex motor skills, [504–505](#)

Compound conditioning, [134–135](#)

Concept

- defined, [249](#)
- discrimination learning of, [250–251](#)

Concept formation, [226–275](#), [249–252](#)

- behavioral processes, [228–255](#)
- brain substrates, [255–266](#)
- category learning and prototypes, [249–252](#)
- clinical perspectives, [266–273](#)
- cortical representations and generalization, [257–263](#)
- defined, [228](#)
- discrimination learning and stimulus control, [238–243](#)
- emergence in concepts through discrimination learning, [250–251](#)
- prototypes and structure of natural categories, [251–252](#)
- similarity and dissimilar stimuli for predicting same outcome, [243–249](#)

Concurrent reinforcement schedules, [198](#)

Concussion, [319](#)

Conditioned avoidance, [430–432](#), [431f](#)

Conditioned compensatory response, [131–132](#)

Conditioned emotional responses, [126–127](#), [428–434](#), [488](#)

- extinction and, [430](#), [458–459](#)

Conditioned escape, [430](#)

Conditioned response (CR), [123–125](#)

- extinction of, [168–169](#)

Conditioned stimulus (CS), [123–125](#)

- cues for, [132–133](#)
- hippocampus in, [160–161](#)
- modulation theory for, [146–149](#)
- sex as, [132–133](#), [133tt](#)

Conditioned taste aversion, [151–152](#)

Conditioning. *See also* [Classical conditioning](#); [Instrumental conditioning](#); [Operant conditioning](#)

- appetitive, [125–126](#)
- aversive, [126–127](#)

- delay, [150](#)
- eyeblick, [127–131](#)
- observational, [475–477](#)
- operant, [6–7](#), [8](#), [476](#)
- trace, [150–151](#)
- Conditioning experiments, [131–132](#), [134](#), [232–233](#), [449](#)
- Confabulation, [310–311](#)
- Confirmation bias, [253](#)
- Connectionist models, [28–30](#), [145](#), [149](#), [235](#)
- Consequential region, [231](#)
- Consolidation period, [296–297](#)
- Constant practice, [337](#)
- Constraint-induced movement therapy, [111–112](#), [112f](#)
- Contiguity
 - association, principle of, [1–2](#), [135](#)
 - blocking effect and, [138](#)
 - defined, [1](#), [135](#)
 - learning frequency and, interaction between, [1–2](#)
 - spatial, [1–2](#)
 - temporal, [1–2](#), [190–191](#)
- Continuous reinforcement schedules, [194](#)
- Controlled updating of short-term memory buffers, [381–384](#)
- Co-occurrence of stimuli, [244–245](#)
- Copying
 - defined, [469](#)
 - emulation and goals of, [474–475](#)
 - matching actions without true imitation, [475–478](#)
 - social learning theory and, [470–472](#)
 - studies of true imitation, [472–474](#)
 - vocal imitation and, [479–480](#)
 - vocalizing without imitating, [480–481](#)
 - what is heard, [478–483](#)
 - what is seen, [469–478](#)
- Cortex. *See also* [Prefrontal cortex \(PFC\)](#)
 - activity, [58–61](#), [58f](#), [66f](#)
 - association, [301–302](#)

- defined, [38](#)
- dorsal anterior cingulate, [214f](#), [215](#)
- expansion of, [355–356](#)
- exposure, reorganization after, [105–106](#), [105f](#)
- frontal, [309–310](#)
- generalization and representations, [257–263](#)
- insular (insula), [214](#), [214f](#)
- olfactory, [302](#)
- orbitofrontal, [206–208](#)
- plasticity of, [103–105](#), [261–263](#)
- representations of skills in, [354–358](#)
- sensory, [302](#)
- sensory stimuli, [257–260](#)
- shared-elements models of receptive fields, [258–260](#)
- somatosensory, [102–103](#), [103f](#)
- storage of skill memory, [356–358](#), [357f](#)
- topographic organization in generalization, [260–261](#)
- visual, [302](#), [302f](#)

Cortisol, [420](#)

Cued recall, [289](#)

Cumulative recorder, [179–181](#)

D

- Danger, [430–432](#), [431f](#)
- Darwin, Charles, [6](#), [15–17](#), [17f](#), [31t](#)
- Darwin's theory of evolution, [15–17](#)
- Data, [1](#)
- Declaration of Independence, [11](#)
- Declarative memory, [280](#)
- Deep brain stimulation, [363](#), [363f](#)
- Delay conditioning, [150](#), [505](#)
- Delay discounting, [201–202](#), [201f](#)
- Delayed non-match-to-sample (DNMS) task, [377–378](#)
- Delayed reinforcement, [201–202](#), [207–208](#), [208f](#)
 - in addiction treatment, [220](#)
- Delayed-response eye-gaze task, [395–397](#), [396–397f](#)
- Delay forms of eyeblink conditioning, [150–151](#), [150f](#)
- Dementia, [541](#). *See also* [Alzheimer's disease](#)
- Dendrite, [41](#), [42f](#)
- Dependent variable, [6](#), [7f](#)
- Depression
 - learned helplessness and, [433–434](#)
 - sensitization to, [109–110](#)
- Descartes, René, [9–10](#), [9f](#), [10f](#), [31t](#)
- Development and aging, [500–550](#)
 - adolescence, [512–515](#)
 - Alzheimer's disease, [541–547](#)
 - behavioral processes and, [502–520](#)
 - brain changes from adulthood to old age, [533–538](#)
 - brain changes in adolescence, [529–533](#)
 - brain substrates of, [520–538](#)
 - clinical perspectives on, [538–547](#)
- Down syndrome, [539–541](#)
 - genetic basis of learning and memory, [520–524](#)
 - memory development from infancy through childhood, [502–508](#)

- memory from adulthood through old age, [515–519](#)
- neurons and synapses in brain development, [526–528](#)
- sensitive periods for early learning, [508–512](#)
- Difference image, [52](#), [56](#)
- Differential reinforcement of alternative behaviors (DRA), [189–190](#), [220](#)
- Diffusion tensor imaging (DTI), [40](#)
- Digit span, [27](#), [513](#)
- Directed forgetting, [292](#), [292f](#), [309](#), [309f](#), [322](#), [457–458](#)
- Direct-matching hypothesis, [489](#)
- Discrete-component representation, [232](#)
- Discrete trials paradigm, [178–179](#)
- Discrimination, [252–254](#)
- Discrimination learning, [227–275](#)
 - behavioral processes, [228–255](#)
 - brain substrates, [255–266](#)
 - clinical perspectives, [266–273](#)
 - concept formation, category learning and prototypes, [249–252](#)
 - of concepts, [250–251](#)
 - cortical representations and generalization, [257–263](#)
 - defined, [227](#)
 - discrimination learning and stimulus control, [238–243](#)
 - errorless, and easy-to-hard transfer, [242–243](#)
 - and learned specificity, [239–242](#)
 - similarity and dissimilar stimuli for predicting same outcome, [243–249](#)
 - stimulus control and, [238–243](#)
- Discriminative stimulus, [177](#), [181](#)
- Dishabituation, [79](#), [80](#)
- Dissociative amnesia, [321–322](#)
- Dissociative fugue, [321–322](#)
- Distancing, [220](#)
- Distributed representation, [29](#), [29f](#), [235–238](#), [247](#), [258–259](#)
- DNA, [520](#). *See also* [Genetics](#)
- Dog salivation, [123](#), [125](#)
- Dolphins
 - skill memory in, [333–334](#)
 - social learning and memory in, [467](#)

Dopamine, [68](#), [210–212](#), [408–409](#), [529–530](#)

Dorsal anterior cingulate cortex (dACC), [214f](#), [215](#)

Dorsal posterior insula, [214](#)

Dorsal striatum, [205–206](#), [205f](#), [349](#)

Dorsolateral prefrontal cortex (DLPFC), [394](#), [394f](#)

- cognitive control and, [399–400](#)
- in working memory, [395–400](#)

Double-blind design, [21](#)

Down syndrome, [538–541](#)

- Alzheimer’s disease and, connection between, [545–546](#)
- animal models of, [540–541](#)
- brain abnormalities and memory impairments in, [539–540](#)

Drive reduction theory, [185](#)

Drug addiction

- classical conditioning in, [216–218](#), [217f](#)
- classical conditioning in tolerance to, [166–168](#)
- extinguishing, [169](#)
- treatments for, [219–221](#)

Drugs, [68–70](#)

- addiction. *See* [Drug addiction](#)
- dogs in detecting, [184](#)
- tolerance study on, [167](#), [167f](#), [168](#)

Dualism, [9–10](#)

Dual process theory, [84](#), [85f](#), [91](#)

Dual-task experiment, [376–377](#)

Dysexecutive syndrome, [393](#)

E

Easy-to-hard transfer, [242–243](#)

Ebbinghaus, Hermann, [19–23](#), [20f](#), [291](#)

Echolalia, [492](#)

Ecological constraints, [152](#)

Economics of behavior, [199–201](#), [200f](#)

Electroconvulsive shock, [296–297](#), [297f](#)

Electrodermal activity (EDA), [82](#), [83f](#)

Electroencephalography (EEG), [52–53](#), [52f](#)

Electromagnetic control of neurons, [65–68](#)

Electrophysiological experiment, [259–260](#), [260f](#)

Electrophysiological recordings in cerebellum, [155–156](#), [156f](#)

Elicited imitation, [506](#)

Emotion, [418–424](#), [434–438](#). *See also* [Emotional influences on learning and memory](#)

 amygdala in, [440–445](#)

 autonomic arousal and fight-or-flight response, [420–421](#), [421t](#)

 Cannon–Bard theory of, [421f](#), [422](#)

 conscious feelings and, [446–447](#)

 defined, [418](#)

 fear response across species, [425–426](#)

 hippocampus, encoding in, [449–450](#)

 James–Lange theory of, [421–422](#), [421f](#)

 learning pathways in amygdala, [442–443](#)

 memory encoding and, [434–435](#)

 memory retrieval and, [435–436](#)

 memory storage and retrieval, impact on, [434–438](#)

 in nonhuman animals, [424–428](#)

 opponent process theory and, [85–86](#)

 other than fear, [427–428](#)

 theories of, [421–424](#), [421f](#)

Emotional contagion, [475](#)

Emotional influences on learning and memory, [416–465](#). *See also* [Emotion](#)

 across the lifespan, [518](#), [518f](#)

- amygdala in, [440–445](#)
- behavioral processes and, [418–439](#)
- brain substrates and, [439–453](#)
- clinical perspectives on, [453–461](#)
- conditioned responses to fear, [428–434](#)
- conscious feelings and frontal lobes, [446–447](#)
- encoding emotions in hippocampus, [449–450](#)
- phobias, [453–461](#)
- posttraumatic stress disorder, [456–461](#)
- Emotional response expression, [441–445](#)
- Emotional responses, conditioned, [428–430](#), [488](#)
- Empiricism, [2–3](#), [31t](#)
- Emulation, [472](#)
- Encoding specificity effect, [287–288](#)
- Endogenous opioids, [68–69](#), [212–213](#)
- Engram, [59](#), [304](#)
- Enriched environment, [44](#), [44f](#)
- Environment, genetic blueprint influenced by, [520–526](#)
- Environmental enrichment, [44](#), [44f](#)
- E.P. (amnesic patient)
 - anterograde amnesia in, [305](#), [312](#), [318](#)
 - bilateral medial temporal damage in, [305](#), [312](#)
 - episodic memories and, [305](#)
 - memory tests of, [305](#), [306f](#)
 - retrograde amnesia in, [312](#)
 - semantic memory in, [308](#)
- Epigenetics, [524–526](#)
- Epilepsy, [271–272](#)
- Epinephrine, [420](#), [449](#)
- Episodic memory
 - behavioral processes and, [278–301](#)
 - brain substrates and, [301–318](#)
 - clinical perspectives on, [319–323](#)
 - defined, [278](#)
 - determining what gets stored in, [309–311](#)
 - distinguishing between, [280–281](#)

- features of, [279–284](#)
- frontal cortex in memory storage and retrieval, [309–310](#)
- functional amnesia, [321–323](#)
- hippocampus and forming new, [305–307](#)
- medial temporal lobes in memory storage, [304–309](#)
- memory consolidation and reconsolidation, [296–298](#)
- memory encoding, [284–287](#)
- memory failure, [291–296](#)
- metamemory, [298–300](#)
- neuronal networks for semantic memory, [301–304](#)
- nonhuman, [282–284](#), [307–308](#)
- old memories vs. new learning and, [517–518](#)
- retrieving existing memories, [287–291](#)
- sequence of, [281–282](#)
- storage of, [445–446](#)
- transient global amnesia, [320–321](#)

Equipotentiality, [59](#)

Equivalence, acquired, [245–246](#)

Error correction

- associative weights and compound conditioning, [140](#)
- classical conditioning, [135–146](#)
- error-correction learning, [139–140](#)
- in human category learning, [143–146](#)
- informational value of cues, [135–136](#)
- Kamin’s blocking effect, [136–138](#)
- and modulation of unconditioned stimulus, [148](#)
- in Rescorla–Wagner model, [138–146](#), [140t](#)

Error-correction learning, [139–140](#)

Errorless discrimination learning, [242–243](#)

Escape conditioning, [430](#)

Estes, W. K., [24–26](#), [26f](#), [31t](#)

Estrogen, [515](#), [530–531](#)

Event-related potential (ERP), [53](#)

Evolution, [332](#)

- Darwin’s theory of, [15–17](#), [425](#)
- defined, [15](#)

- fear responses and, [425](#)
 - by natural selection, [15](#)
- Executive control, [381](#)
- Executive processing, [398–403](#)
- Experiment
 - Bandura's Bobo doll, [469–470](#), [486](#)
 - blocking, [137–138](#), [140](#), [141–142](#)
 - conditioning, [134](#), [232–233](#), [449](#)
 - dual-task, [376–377](#)
 - electrophysiological, [259–260](#), [260f](#)
 - episodic vs. semantic memory, [280–281](#)
 - free-operant, [179–180](#), [180f](#)
 - generalization, [229–230](#)
 - memory, [293](#)
 - Pavlov's, [125](#)
 - sexual habituation, [80](#)
 - in social manipulation of behavior, [486–487](#)
- Experimental psychology, [4](#), [12–14](#), [19–24](#)
 - human memory experiments, [19–21](#)
 - law of effect and, [8](#)
- Experimenter bias, [21](#)
- Expert, [329](#)
- Expertise, acquiring, [339–341](#)
- Explicit learning, [339](#)
- Explicit memory, [280](#)
- Exploration, learning by, [93–95](#), [93f](#)
- Exposure, cortical reorganization after, [105–106](#), [105f](#)
- Exposure-based learning, [86–95](#)
 - novel object recognition and, [87–88](#)
 - perceptual learning and, [89–92](#)
 - priming and, [88–89](#), [88f](#)
 - spatial learning and, [93–95](#)
- Extinction, [133–134](#)
 - avoidance learning and, theories of, [432](#)
 - classical conditioning and, [177–180](#)
 - conditioned emotional responses and, [430](#), [458–459](#)

- of conditioned responses, [168–169](#)
- defined, [7](#), [133](#)
- of eyeblink conditioning, [132f](#), [133](#), [133f](#)
- operant conditioning and, [177–181](#)
- posttraumatic stress disorder and, [458–459](#)

Extradimensional discrimination, [240](#)

Eyeblink conditioning, [127–131](#)

- acquisition of, [131f](#), [133](#)
- classical conditioning and, [127–131](#), [178](#)
- delay and trace forms of, [150–151](#), [150f](#)
- extinction of, [133](#), [133f](#)
- face-slap, [127f](#)
- hippocampal region in, [263–264](#), [264f](#)
- in humans, [128f](#)
- latent inhibition in, [147](#), [147f](#)
- learning curves for, [130–131](#), [132f](#)
- negative patterning in, [246–247](#)
- in rabbits, [128–129](#), [128f](#), [129f](#)

F

Face-slap eyeblink conditioning, [127f](#)

False memory, [56](#), [294–296](#), [324](#)

Familiarity, [87](#)

Familiarization

- behavioral processes, [75–119](#)

- brain substrates, [96–110](#)

- clinical perspectives, [110–116](#)

- defined, [87](#)

- exposure-based learning, [86–95](#)

- habituation, [111–112](#)

- human–machine interfaces, [114–116](#)

- invertebrate model system, [96–101](#)

- perceptual learning and cortical plasticity, [101–106](#)

- recognizing and responding to repetition, [77–86](#)

- rehabilitation after stroke, [111–112](#)

- sensitization to stress in anxiety and depression, [112–114](#)

- sensory modality regaining through perceptual learning, [114–116](#)

- temporal lobe involvement in spatial learning and familiarity, [106–110](#)

Faster forgetting, [36](#)

Fear, [424–426](#)

- conditioned avoidance and learning to avoid danger, [430–432](#), [431f](#)

- conditioned emotional responses and learning to predict danger, [428–434](#)

- conditioned escape and learning to get away from danger, [430](#)

- learned helplessness, [432–434](#), [432f](#)

- response to, [418](#)

Feeling of knowing (FOK), [299](#), [519](#)

Feelings, frontal lobes and, [446–447](#). *See also* [Emotion](#)

Fetal magnetoencephalography (fMEG), [503](#)

Fight-or-flight response, [420–421](#), [420t](#)

- tend-and-befriend vs., [426–427](#)

Filtering, collaborative, [234](#)

Finches, [16](#), [16f](#)

Fixation time, [78](#)

Fixed-interval (FI) schedule, [195–196](#), [195f](#)

Fixed-ratio (FR) schedule, [194–195](#), [195f](#)

Flashbulb memories, [436–437](#)

Food preferences, [484–485](#)

Forebrain, basal, [310–311](#)

Forgetting, [291–292](#), [345–347](#)

- defined, [20](#)
- directed, [292](#), [309](#), [309f](#), [322](#), [457–458](#)
- intentional, [292](#), [309–310](#), [457–458](#)
- memory failure of, [292](#), [345–347](#)
- passive, [291–292](#), [292f](#), [296](#), [300](#), [346](#)
- retention curve for, [20](#)
- simple, [291–292](#)
- tips for faster, [36](#)

Fornix, [310](#)

Free-operant experiment, [179–180](#), [180f](#)

Free-operant paradigm, [178–181](#)

Free recall, [289](#)

Freezing, [429–430](#)

Frontal brain activity during working-memory tasks, [395–398](#)

Frontal cortex, [309–310](#)

Frontal-lobe damage, [391–395](#)

Frontal lobes, [38](#)

- behavior changes following damage to, [392–393](#)
- comparative anatomy of, [392f](#)
- conscious feelings and, [446–447](#)
- damage to, memory and, [391–395](#)
- organization of, goal abstraction and, [402–403](#)
- prefrontal cortex divisions, [394–395](#), [394f](#)
- working memory deficits following damage to, [393–394](#)

Functional amnesia, [321–323](#)

Functional magnetic resonance imaging (fMRI), [52](#), [53](#)

Functional neuroimaging, [51–52](#), [52f](#)

Functional properties of learning and memory, [47–57](#)

- brain functions, [47–55](#)

neuron recording, [53–55](#), [54f](#)

G

Galápagos Islands, [16](#), [16f](#)

Garcia–Koelling taste-aversion study, [151–152](#), [151t](#)

Gender differences

in cognitive skills, [343](#)

in learning and memory, [514–515](#)

Generalization, [7](#), [227–275](#)

acquired equivalence in, [267–269](#)

auditory, [258–262](#)

autism and, [270–272](#)

behavioral processes, [228–255](#)

brain substrates, [255–266](#)

challenges of incorporating similarity in learning models, [231–234](#)

clinical perspectives, [266–272](#)

concept formation, category learning and prototypes, [249–252](#)

cortical representations and, [257–263](#)

defined, [227](#)

discriminating between paradigms of, [248](#)

discrimination learning and stimulus control, [238–243](#)

hippocampal region in, [263–266](#)

hippocampus and, [70–71](#), [268–269](#)

of knowledge and skills, [249](#)

meaning-based, [246–248](#)

peak shifts in, [229–230](#)

schizophrenia, [267–270](#)

as search for similar consequences, [230–231](#)

sensory stimuli, [257–263](#)

shared elements and distributed representations, [235–238](#)

similarity and dissimilar stimuli for predicting same outcome, [243–247](#)

similarity-based, [246–248](#)

stereotypes, discrimination, racism and, [252–254](#), [252–255](#)

and stimuli prediction with similar outcomes, [229–238](#)

stimulus, [229–238](#), [244–245](#)

- stimulus specificity and, [79](#)
- topographic organization of, [260–261](#)

Generalization experiment, [229–230](#)

Generalization gradient, [229–230](#)

Genes, [520](#). *See also* [Genetics](#)

Genetics

- of Alzheimer’s disease, [544–545](#)
- epigenetics, [524–526](#)
- individual differences in learning abilities and, [520–521](#)
- influence of experience on, [520–526](#)
- of learning and memory, [520–524](#)
- of schizophrenia, [408–409](#), [409f](#), [410f](#)
- selective breeding and twin studies, [521–523](#), [522f](#)
- in song learning, [481–483](#)

Gestational age, [502](#)

Glia, [41–42](#)

Global abstraction, [402–403](#)

Glucocorticoid, [420](#), [450](#)

Goals

- abstraction of, frontal-lobe organization and, [402–403](#)
- setting and planning, [384–385](#)

H

Habit slip, [181](#), [432](#)

Habituation, [78f](#), [111–112](#)

in *Aplysia*, [96–101](#), [162t](#)

behavioral processes, [76–95](#)

brain substrates, [96–110](#)

clinical perspectives, [110–116](#)

cortical plasticity, [101–106](#)

defined, [77](#)

dual process theory of, [84](#), [85f](#), [91](#)

exposure-based learning, [86–95](#)

human–machine interfaces, [114–116](#)

invertebrate model system, [96–101](#)

in learning before birth, [502–503](#), [503f](#)

maximizing, [86](#)

perceptual learning vs., [92](#), [101–106](#)

recognizing and responding to repetition, [77–86](#)

rehabilitation after stroke, [111–112](#)

sensitization to stress in anxiety and depression, [112–114](#)

sensory modality regaining through perceptual learning, [114–116](#)

temporal lobe involvement in spatial learning and familiarity, [106–110](#)

Hebbian learning, [45–46](#), [45f](#)

Hedonic value, [209–210](#)

Heterosynaptic, [100](#)

High Bridge study, [423](#)

Highly superior autobiographical memory (HSAM), [55–56](#)

High vocal center (HVC), [491](#)

Hippocampal region, [263–266](#)

Hippocampus, [39](#), [39f](#), [45](#)

in animals, [106–107](#), [107f](#), [160f](#)

classical conditioning and, [160–161](#), [263–264](#), [304](#), [449](#)

defined, [304](#)

emotional influences on memory and, [439](#)

- emotions, encoding in, [434–435](#), [448–450](#)
- encoding of socially transmitted food preferences, [484–485](#)
- familiar object recognition, [109–110](#)
- generalization and, [263–266](#)
- in humans, [160f](#)
- place identification, [107–109](#)
- in spatial learning and familiarity, [106–110](#)

H.M. (amnesic patient)

- anterograde amnesia in, [305](#), [308](#), [318](#)
- implicit memory in, [280](#)
- medial temporal lobes, studied, [304–305](#)
- memory tests of, [305](#)
- semantic memory in, [308](#)
- story of, [277–278](#)

Homeostasis, [132](#)

Homosynaptic, [99](#)

Homunculus, [66–67](#), [66f](#), [103f](#), [258](#), [502](#)

Hornet magazine, [17f](#)

Hull, Clark, [23–24](#), [24f](#), [31t](#)

Human

- brain, [38–39](#), [39f](#)
- category learning, [143–146](#)
- eyeblick conditioning in, [127f](#), [128f](#)
- hippocampus in, [160f](#)
- memory experiments, [19–21](#)
- motor cortex, [65–66](#), [66f](#)

Human–machine interfaces, learning to control, [363–365](#)

Hyperthymesia, [55](#)

Hypothalamus, emotional influences on memory and, [439](#)

Hypothesis, [21](#)

I

- Identical elements theory, [344](#)
- Image/imaging difference, [52](#), [56](#)
- Imitation, elicited, [506](#)
- Imitative deficits after stroke, [492–493](#), [493f](#)
- Implicit learning, [338–339](#)
- Implicit memory, [280](#)
- Imprinting, [508–509](#)
- Incentive salience hypothesis, [211–212](#), [211f](#)
- Incoming stimuli, [49](#), [49f](#)
- Independent variable, [6](#), [7f](#)
- Individual differences in learning abilities, [520–521](#)
- Inductive inference, [251–252](#)
- Inferior olive, [155](#), [157f](#)
- Informational redundancy and learning, [135t](#)
- Informational value of cues, [135–136](#)
- Information theory, [26–28](#)
- Inhibitory feedback, [159–160](#)
- Injury effects on brain, [35](#), [58](#), [59](#)
- Instrumental conditioning, [176](#). *See also* [Operant conditioning](#)
- Insular cortex (insula), [214](#), [214f](#)
- Intelligence
 - classical music and, [525](#)
 - as key to working memory, [388–389](#)
- Intentional forgetting, [292](#), [309](#), [457–458](#)
- Interference, [293–294](#)
- Internet gaming disorder (IGD), [219](#)
- Interneurons, [41](#)
- Interpositus nucleus, [154–155](#)
- Interstimulus interval (ISI), [151](#)
- Intradimensional discrimination, [240](#)
- Invertebrates
 - and cellular basis of learning, [161–165](#)

classical conditioning, [161–165](#)

habituation in *Aplysia*, [96–101](#)

habituation model system, [96–101](#)

long-term structural changes and creation of new synapses, [164–165](#)

presynaptic vs. postsynaptic changes during learning, [163–164](#)

sensitization in sea hares, [99–101](#)

J

James, William, [3–4](#), [3f](#), [8](#), [31t](#)

James–Lange theory of emotion, [421–422](#), [421f](#)

James memory model, [4](#), [4f](#), [6](#)

Judgment of learning (JOL) phenomenon, [299–300](#)

K

Kamin's blocking effect, [136–138](#), [137t](#)

Karaoke, [480](#)

Knowledge of results, [334](#)

L

Landmarks, [94f](#)

use of by wasps, [94–95](#), [94f](#)

visual, [107–108](#), [108f](#)

Land mine detection, [175](#), [176](#), [176f](#)

Language learning, [510–512](#)

Latent inhibition, [146–147](#), [147t](#), [264](#), [264f](#)

in eyeblink conditioning, [147f](#)

Latent learning, [18](#), [76–77](#), [86](#), [93](#), [95](#), [96](#), [106](#), [109](#)

Law of effect, [8](#), [176–177](#), [187–188](#), [235](#), [470](#)

Learned association, [181–190](#)

chaining, [183](#)

discriminative stimuli, [181](#)

punishers, [187–189](#)

reinforcers, [183–187](#)

responses, [182](#)

shaping, [182–183](#)

Learned helplessness, [432–434](#), [432f](#)

Learned non-use, [111–112](#), [112f](#)

Learned specificity, [239–242](#)

Learning. *See also* [Learning and memory](#); *specific types of*

abilities for, [520–521](#)

about emotional responses, [428–434](#)

avoidance, theories of, [431–432](#), [431f](#)

before birth, [502–504](#)

brain functional properties in, [47–57](#)

brain structure changes from, [43–46](#)

category, [145f](#), [249–252](#)

cognitive skill, [353–354](#)

curve, [6–7](#), [178](#)

deficits after lesions, [349–351](#)

defined, [2–3](#)

discrimination, [227](#)

error-correction, [139–140](#), [140t](#)
explicit, [339](#)
by exploration, [93–95](#), [93f](#)
exposure-based, [86–95](#)
frequency and, interaction between, [1–2](#)
genetic variation in abilities, [520–521](#)
Hebbian, [45–46](#), [45f](#)
hippocampal region in, [264–266](#)
human category, [143–146](#)
implicit, [338–339](#)
informational redundancy and, [135–136](#), [135t](#)
invertebrates and cellular basis of, [161–165](#)
language, [510–512](#)
latent, [18](#), [76–77](#), [86](#), [93](#), [95](#), [96](#), [106](#), [109](#)
mathematical models of, [23–24](#)
mere exposure, [89–91](#)
new associations, [130–131](#), [132f](#)
in old age, [519](#)
pathways in amygdala, [442–443](#)
perceptual, [89–92](#)
set formation, [345](#)
spatial, [93–95](#)
statistical, [90](#)
varieties of, [162t](#)
while you sleep, [317–318](#)

Learning and memory. *See also* [Memory](#)

associationism associated with, [1–3](#), [6–8](#)
behaviorism associated with, [11–14](#), [17–19](#)
cognitive approach to, [30](#)
dualism associated with, [9–10](#)
empiricism associated with, [2–3](#), [6–7](#), [31t](#)
linking ideas in the mind in, [3–9](#)
male–female differences in, [514–515](#)
nature and nurture’s role in, [9–14](#)
philosophy of, [3–9](#)
psychology of, [3–14](#)

theory of natural selection associated with, [16–17](#), [17t](#)
Who's Who in the history of, [23](#)

Learning curve, [6–7](#), [7f](#)

Learning set formation, [345](#)

Lesion, [40](#)
brain, [40](#), [59](#), [350](#)
learning deficits after, [349–351](#)

Levels-of-processing effect, [286–287](#)

Limbic system, [439](#)

Localized neuron and synapse loss, [533–535](#), [534f](#)

Locke, John, [10–11](#), [10f](#), [31t](#)

Long-term depression (LTD), [64–65](#)

Long-term memory, [370](#)
behavioral distinctions between short-term memory and, [372t](#)

Long-term potentiation (LTP), [63–65](#), [63f](#), [444](#), [520–521](#)

Long-term structural changes, [164–165](#)

M

Mackintosh model of classical conditioning, [149](#), [150](#)

Magnetic resonance imaging (MRI), [40](#), [41f](#)

Maintenance (rehearsal), [399–400](#)

Male–female differences in learning and memory, [514–515](#)

Mammalian conditioning of motor reflexes, [127–131](#), [153–161](#)

- brain stimulation as substitute for behavioral training, [157–158](#)

- classical conditioning, [153–161](#)

- electrophysiological recording in cerebellum, [155–156](#)

- error correction through inhibitory feedback, [159–160](#)

- hippocampus in conditioned stimulus (CS), [160–161](#)

- impaired conditioning following cerebellar damage, [158](#)

Manipulation (cognitive control), [399–400](#)

Map/mapping

- cognitive, [17–19](#), [19f](#), [109](#)

- executive processing and working memory onto prefrontal cortex, [398–403](#)

- global abstraction and frontal-lobe organization, [402–403](#)

- maintenance (rehearsal) vs. manipulation (cognitive control), [399–400](#)

- neural bases of state-based accounts of working memory, [401](#), [401f](#)

- visuospatial and phonological-verbal working memory, [399–400](#)

Massed exposure, [80](#)

Massed practice, [336](#)

Matching law of choice behavior, [198–199](#), [198f](#)

Mathematical models of learning, [23–24](#)

Mathematical psychology, [24–26](#), [126](#)

Medial septum, [310](#)

Medial temporal lobes, [263](#), [263f](#), [277–278](#), [304–309](#). *See also* [Hippocampus](#)

Media's impact on behavior, [486–488](#), [487f](#)

Medication reduction, [168–169](#)

Memory. *See also* [Aging memory](#); [Learning and memory](#); *specific types of*

- brain, impairments in Down syndrome, [539–540](#)

- concussion and, [319](#)

- consolidation and reconsolidation, [296–298](#), [313](#)

- declarative, [280](#)
- defined, [2](#)
- determining what gets stored, [309–311](#)
- of emotional events, [434–438](#)
- explicit, [280](#)
- extraordinary memorizers, [291](#)
- false, [294–296](#), [324](#)
- flashbulb, [436–437](#)
- hippocampus and cortex interaction during consolidation, [313–315](#)
- implicit, [280](#)
- long-term, [370](#)
- mood congruency of, [435–436](#)
- nondeclarative, [330](#)
- pill for improving, [70](#)
- sensory, [371–372](#)
- short-term, [370](#), [381–384](#)
- skill, [327–367](#)
- sleep and, [315–318](#)
- study habits and, [300](#)
- tips for better, [5](#)
- transient, [370–374](#)

Memory development from infancy through childhood, [502–508](#)

- conditioning and skill learning in young children, [504–506](#)
- episodic and semantic memory development, [506–508](#)
- learning before birth, [502–504](#)

Memory encoding, [284–287](#)

- conditions at, retrieval and, [287–288](#)
- deeper processing for, [286–287](#)
- emotion and, [434–435](#)
- exposure does not guarantee, [285](#)
- practice and expertise in, [339–341](#)
- prior knowledge and sustaining, [285–286](#)
- skill memory, [334–343](#)
 - repetition and, [334–336](#)
- specificity effect in, [287–288](#)
- talent in, [341–342](#)

- timing and sequence for, [336–337](#)
- Memory failure, [291–296](#), [345–347](#)
 - false memories, [294–296](#)
 - forgetting, [291–292](#), [292f](#), [345–347](#)
 - interference, [293–294](#)
 - source monitoring, [294](#)
- Memory reconsolidation, [451–452](#)
- Memory retrieval, [287–291](#), [344–347](#)
 - amygdala, [450–452](#)
 - brain activity in remembering, [55–57](#), [56f](#)
 - cues for, [289](#)
 - emotion and, [435–436](#)
 - encoding conditions and, [287–288](#)
 - failure at, and improved memory, [289–291](#)
 - stress and, [450–452](#)
 - struggling in, and improved memory, [289–291](#)
- Mere exposure learning, [89–91](#)
- Met allele* (of *BDNF* gene), [521](#)
- Metamemory, [298–300](#), [310](#), [518–519](#)
- Method of loci, [291](#)
- Methylation, [524–525](#)
- Miller, George, [26–28](#), [27f](#), [31t](#)
- Mind palace, [291](#)
- Mirror neurons, [489–492](#)
- Mirror reading, [360](#)
- Mirror tracing, [359–360](#), [360f](#)
- Mnemonics, [55](#), [291](#)
- Modulation theory
 - for conditioned stimulus, [146–147](#), [149](#)
 - unconditioned stimulus, [148](#)
- Mood congruency of memory, [435–436](#)
- Mother memory module, [43–44](#)
- Motivational value of a stimulus, [209–210](#)
- Motor control, [49–51](#), [49f](#), [50f](#)
- Motor cortex, [65–66](#), [66f](#)
- Motor program, [339–340](#)

Motor prosthesis, [363–364](#)

Motor reflex conditioning, [154f](#), [160](#)

Motor reflexes

- brain stimulation as substitute for behavioral training, [157–158](#)

- classical conditioning, [153–161](#)

- electrophysiological recording in cerebellum, [155–156](#)

- error correction through inhibitory feedback, [159–160](#)

- hippocampus in conditioned stimulus, [160–161](#)

- impaired conditioning following cerebellar damage, [158](#)

- mammalian conditioning of, [127–131](#), [153–161](#)

Mozart effect, [525](#)

Multiple trace theory, [313–314](#)

Multitasking, [36](#)

Musician's dystonia, [356](#)

Myelin, [42](#)

N

Naltrexone, [220](#)

Nativism, [2](#), [11](#)

Natural history, [6](#), [15–16](#), [30](#), [31t](#)

Natural sciences for explaining mind, [28](#)

Natural selection, [15–17](#), [26](#), [43](#)

 evolution by, [15](#)

Nature and nurture's role in learning and memory, [9–14](#)

Negative contrast, [185](#)

Negative patterning, [246–248](#)

Negative punishment, [192](#)

Negative reinforcement, [191–192](#), [192t](#)

Neo-behaviorism, [17–19](#)

Neo-Hullians, [24](#)

Neophobia, [87](#)

Nervous system, [37](#)

 activity manipulation, [65–68](#)

 brain, [37–46](#)

 brain function research, [65–70](#)

 central, [37](#), [37f](#)

 experience and changes in brain structure, [43–46](#)

 neural connection changes, [45–46](#)

 peripheral, [37](#), [37f](#)

 structural properties, [36–46](#), [37f](#)

Neural activity during perceptual-motor skill learning, [351–353](#)

Neural bases of state-based accounts of working memory, [401](#), [401f](#)

Neural connection changes, [65–70](#)

 Hebbian learning, [45–46](#)

 long-term depression, [64–65](#)

 long-term potentiation, [63–65](#), [63f](#)

Neural connectivity, [45](#)

Neuroanatomy of brain, [38–42](#), [38f](#)

Neurofibrillary tangle, [542](#), [542f](#)

Neurogenesis, [526](#), [533–535](#), [534f](#), [536–538](#)

Neuroimaging, functional, [51–52](#), [52f](#)

Neuromodulator, [62](#)

Neuronal wiring, [528](#)

Neurons, [37](#)

- in aging brains, [536–538](#)
- auditory cortex in receptive field of, [102–103](#), [102f](#)
- in brain, [41–42](#), [42f](#)
- connections, [45–46](#), [45f](#), [60–62](#)
- early overproduction of, [526–527](#)
- imaging, [49](#)
- mirror, [489–492](#)
- networks for semantic memory, [301–304](#)
- pruning of synapses, [527–528](#)
- recording, [53–55](#), [54f](#)
- selective response of and memory, [302–303](#)
- sensitive periods of neuronal wiring, [528](#)
- and synapse loss, [533–535](#), [534f](#)
- synapses, [59–62](#), [61f](#)
- and synapses in brain development, [527–528](#)

Neurophysiology, [53](#), [59](#)

Neuroscience, defined, [36](#)

Neuroscience of learning and memory, [35–73](#)

- functional properties and, [47–57](#)
- memory finding and manipulation, [57–70](#)
- nervous system structural properties and, [36–46](#)

Neurotransmitter, [61–62](#)

- dopamine, [68](#), [210–212](#), [408–409](#), [529–530](#)
- norepinephrine, [449–450](#)

Nondeclarative memory, [330](#)

Nonhuman animal emotion, [424–428](#)

Nonhuman episodic and semantic memory, [282–284](#), [307–308](#)

Norepinephrine, [449–450](#)

Novel object recognition, [87–88](#), [87f](#)

Nucleus

- central, [440–441](#)

interpositus, [154–155](#)

Nucleus basalis, [262](#)

O

Object recognition

- familiar objects, [109–110](#)

- novel objects, [87–88](#)

Observational conditioning, [475–476](#)

Observational learning, [468](#)

- electroencephalography, [52–53](#)

- functional neuroimaging, [51–52](#)

- of skills, [335](#)

- structural neuroimaging in humans, [43–44](#)

Occipital lobe, [38](#)

Old age

- brain from adulthood to, [533–538](#)

- localized neuron and synapse loss, [533–535](#), [534f](#)

- loss of synaptic stability, [535–536](#)

- memory through, [515–519](#)

- neurogenesis in, [536–538](#)

Oligodendrocytes, [42](#)

On-the-job training, [44–45](#)

Open skills, [331](#), [332](#)

Operant conditioning, [8](#), [174–225](#)

- addiction treatments, [219–221](#)

- altruism and, [202–204](#)

- avoidance learning and, theories of, [431–432](#), [431f](#)

- basal ganglia and, [349](#)

- behavioral addiction, [218–219](#)

- behavioral processes and, [176–204](#)

- brain substrates of, [205–215](#)

- choice behavior, [198–204](#)

- classical conditioning and, [175–176](#), [495](#)

- clinical perspectives on, [216–221](#)

- defined, [8](#), [175–176](#), [205](#)

- discovery of, [176–181](#)

dorsal striatum and, [205–206](#), [205f](#)
drug addiction, [216–218](#), [217f](#)
escape learning and, [430](#)
extinction and, [177–181](#)
free-, [178–181](#)
imprinting and, [43](#)
incentive salience hypothesis and, [211–212](#), [211f](#)
learned association, [181–190](#)
orbitofrontal cortex and learning to predict outcomes, [206–208](#)
paradigms for, [192t](#)
punishment and, [191–193](#), [213–215](#)
reinforcement and, [191–192](#)
reinforcement schedules, [190–197](#)
responses in, [182](#)
skills and, [331](#), [348–349](#)
social learning theory and, [470](#), [476](#), [495](#)
timing and, [190–191](#)

Opiate receptors, [212](#)
Opioids, endogenous, [212–213](#)
Opponent process theory, [85–86](#)
Optogenetics, [444](#)
Orbitofrontal cortex, [206–208](#)
Orienting response, [77–78](#)
Other-race effect, [91](#)
Outcomes, learning to predict, [206–208](#), [207f](#)
Outgoing responses, [49–51](#), [50f](#)
Overshadowing, [134–135](#)

P

Papez circuit, [439–440](#)

Parietal lobe, [38](#)

Parkinson's disease, [219](#), [362–363](#)

Partial reinforcement schedules, [194](#)

Passive forgetting, [291–292](#), [292f](#), [296](#), [300](#), [346](#)

Pathological addiction, [217–218](#)

Pathways into brain, [49](#), [49f](#)

Pavlov, Ivan, [6–7](#), [7f](#), [8](#), [31t](#), [36](#)

Pavlovian conditioning, [6–7](#), [24](#), [121–122](#), [122f](#), [124f](#), [168](#), [170](#), [227–228](#). *See also* [Classical conditioning](#)

defined, [122](#)

terminology of, [124t](#)

Pavlov's experiment, [125](#)

Pavlov's principle of classical conditioning, [122](#), [176–177](#)

Perceptual learning, [89–92](#)

in adults after exposure, [105–106](#)

and cortical plasticity, [101–106](#)

during development, [104–105](#)

habituation vs., [92](#)

imprinting and, [43](#)

Perceptual-motor skills, [330](#), [351–353](#)

Peripheral nervous system (PNS), [37–38](#), [37f](#)

Perseveration, [394](#)

Perspective taking, [474](#)

Philosophy of learning and memory, [3–8](#)

Phobias, [453–456](#)

causes of, [454–455](#)

classical conditioning and, [454–456](#)

defined, [453](#)

treating, [455–456](#)

Phonological loop, [374](#), [375–376](#)

Phonological-verbal working memory, [399–400](#)

Phrenology, [39–40](#), [47](#), [59](#), [60](#)

Physical sciences for explaining mind, [28](#)

Piloerection, [425](#)

Placebo, [21](#), [168](#), [220](#), [450](#)

Place cell, [107–109](#)

Place identification, [107–109](#), [108f](#)

Plaque

- amyloid, [542](#), [542f](#)
- and tangles in brain, [542–544](#)

Plasticity

- cortical, [103–105](#)
- of cortical representations and generalization, [261–263](#)
- defined, [43](#)
- Hebbian learning and, [46](#)
- presynaptic vs. postsynaptic, [163–164](#)
- synaptic, [62](#)

Plato, [1](#), [2](#), [2f](#), [31t](#)

Positive punishment, [191](#), [192t](#)

Positive reinforcement, [191](#), [192t](#)

Post-reinforcement pause, [194–195](#)

Postsynaptic, [61](#)

Posttraumatic stress disorder (PTSD), [456–461](#)

- causes of, [457–458](#)
- classical conditioning and, [457](#)
- defined, [456](#)
- extinction and, [458–459](#)
- predicting and preventing, [459–461](#)
- treatment of, [458–459](#)

Power law of practice, [334–335](#), [357](#)

Practice. *See also* [Repetition](#)

- musician's dystonia from, [356](#)
- power law of, [335](#), [357](#)
- talent and, [339–341](#)
- timing and sequencing of, [336–337](#)

Precommitment, [201–202](#)

Preconditioning, sensory, [244–245](#), [263–264](#), [264f](#)

Prediction error, [138](#)

Prefrontal cortex (PFC), [391](#)

- adolescence/adolescents, [529–533](#)
- control of long-term declarative memory, [403–407](#)
- dorsolateral (DLPFC), [394](#), [394f](#)
- frontal lobes, [394–395](#), [394f](#)
- remembering and, [55–56](#)
- in schizophrenia, [407–409](#)

Prepulse inhibition, [83–84](#)

Presynaptic, [61](#), [163–164](#)

Primary reinforcers, [185](#)

Primary sensory cortices, [49](#)

Priming, [88–89](#), [88f](#)

Proactive interference, [293–294](#)

Progressive memory loss, [542–544](#)

Prosthesis

- learning to control, [363–365](#)
- sensory, [114–115](#), [363](#)

Prototypes, [249–252](#), [251–252](#)

- in categorization, [250](#)
- discrimination learning of, [250–251](#)
- stereotypes, discrimination, racism, and, [252–254](#)
- and structure of natural categories, [251](#)

Psychogenic amnesia. *See* [Functional amnesia](#)

Psychology

- cognitive, [27](#), [145](#), [149](#)
- defined, [3](#)
- experimental, [12–14](#), [19–24](#)
- of learning and memory, themes in, [1–33](#)
- mathematical, [24–26](#), [126](#)

Puberty, [512–513](#)

Punishers, [183–184](#), [187–189](#)

Punishment

- brain signaling in, [213–215](#)
- defined, [184](#)
- negative, [192](#), [192t](#)

positive, [191](#), [192t](#)

reinforcement vs., [193](#)

Purkinje cells, [154–155](#), [156f](#)

Putamen, [205](#)

Pyramidal cells, [41](#)

R

Rabbits, eyeblink conditioning in, [128–129](#), [128f](#), [129f](#), [150](#)

Racism

 generalization and, [252–254](#)

 unconscious racial bias and, [91](#)

Radial arm maze, [282](#), [282f](#)

Radical behaviorism, [13–14](#)

Rapid eye movement (REM) sleep, [315–317](#)

Receptive field, [102](#)

Receptor, [61](#)

Reciprocal altruism, [203](#)

Reciprocity, [202–204](#)

Recognition, [289](#)

Reconsolidation, [297](#)

Reflex, [7f](#), [47–48](#). *See also* [Motor reflexes](#)

 acoustic startle, [77](#), [78f](#)

 neural circuits and, [96–97](#), [98f](#)

 sensitization of, [83f](#)

Reflex arc, [9–10](#), [10f](#), [51](#)

Refractory period, [62](#)

Reinforcement, [177](#)

 brain signaling and, [210–213](#), [211f](#)

 brain substrates and, [205–206](#), [208–213](#)

 defined, [184](#)

 delayed, [201–202](#), [207–208](#)

 dopamine and brain signaling, [210–212](#)

 endogenous opioids and, [212–213](#)

 negative, [191–192](#), [192t](#)

 positive, [191](#), [192t](#)

 punishment vs., [193](#)

 “wanting” and “liking” in, [209–213](#), [209f](#), [211f](#)

Reinforcement schedules, [190–197](#)

 adding and subtracting outcomes, [191–193](#)

- concurrent, [198](#)
- continuous, [194](#)
- defined, [194](#)
- fixed-interval, [195–196](#), [195f](#)
- fixed-ratio, [194–195](#)
- partial, [194](#)
- response not necessary for, [193–194](#)
- timing affects learning, [190–191](#)
- variable-interval, [195f](#), [196–197](#)
- variable-ratio, [195f](#), [196](#)

Reinforcers, [183–187](#)

Remembering, [55–57](#), [56f](#). *See also* [Memory](#)

Repetition

- dual process theory and, [84](#), [85f](#), [91](#)
- factors influencing rate and duration of, [79–81](#)
- habituation process and, [76–77](#)
- memory encoding and, [334–336](#)
- opponent process theory and, [85–86](#)
- recognizing and responding to, [77–86](#)
- sensitization process and, [82–84](#)
- stimulus specificity and dishabituation, [79](#)

Representational similarity analysis (RSA), [303–304](#), [303f](#)

Rescorla–Wagner model of conditioning, [138](#), [140t](#)

- classical conditioning, [138](#), [143](#), [149](#), [150](#), [160](#), [231–232](#)
- for explaining blocking, [140–142](#), [141f](#)
- influence of, [143](#)

Response, [6](#), [182–183](#)

- conditioned (CR), [123–125](#)
- fear, [418](#)
- fight-or-flight, [420–421](#), [420t](#), [426–427](#)
- tend-and-befriend, [426–427](#)

Retention curve, [20](#), [20f](#)

Retroactive interference, [294](#), [459](#)

Retrograde amnesia, [311–312](#)

Retrograde messenger, [64](#)

Reuptake, [62](#)

Reward prediction error, [212](#)

Ribot gradient, [311–312](#)

Rotary pursuit task, [341–342](#), [342f](#)

Rumelhart, David, [28–29](#), [28f](#), [31t](#)

S

Schizophrenia, [267–270](#)

acquired equivalence in, [267–269](#)

dopamine and genetics of, [408–409](#), [409f](#), [410f](#)

inefficient prefrontal cortical systems in, [408](#)

prefrontal cortex in, [407–409](#)

studies of generalization in, [269–270](#)

Sea hares, [96–101](#), [97f](#)

Seattle Longitudinal Study, [515](#), [516f](#)

Secondary reinforcers, [185–187](#)

Seizures, epileptic, [271–272](#)

Selective breeding, [521–523](#), [522f](#)

Self-control, [201–202](#), [207–208](#), [208f](#)

Semantic memory

behavioral processes and, [278–301](#)

brain substrates and, [301–318](#)

clinical perspectives on, [319–323](#)

defined, [279](#)

determining what gets stored in, [309–311](#)

distinguishing between, [280–281](#)

features of, [279–284](#)

frontal cortex in memory storage and retrieval, [309–310](#)

functional amnesia, [321–323](#)

hippocampus and forming new, [308](#)

medial temporal lobes in memory storage, [304–309](#)

memory consolidation and reconsolidation, [296–298](#)

memory encoding, [284–287](#)

memory failure, [291–296](#)

metamemory, [298–300](#)

neuronal networks for, [301–304](#)

nonhuman, [282](#), [307–308](#)

old memories vs. new learning and, [517–518](#)

retrieving existing memories, [287–291](#)

sequence of, [281–282](#)
transient global amnesia, [320–321](#)

Sensitive period

cortical plasticity and, [104](#)
defined, [508](#)
for early learning, [508–512](#)
imprinting, [508–509](#)
language learning, [510–512](#)
for vision and birdsong, [509–510](#)

Sensitization

in *Aplysia*, [96–101](#), [98f](#), [162t](#)
behavioral processes, [76–95](#)
brain substrates, [96–110](#)
clinical perspectives, [110–116](#)
defined, [82](#)
exposure-based learning, [86–95](#)
general features of, [82–84](#)
habituation, [111–112](#)
hippocampal involvement in spatial learning and familiarity, [106–110](#)
human–machine interfaces, [114–116](#)
to information value of cues, [143–144](#), [143f](#)
invertebrate model system, [96–101](#)
perceptual learning and cortical plasticity, [101–106](#)
recognizing and responding to repetition, [77–86](#)
rehabilitation after stroke, [111–112](#)
in sea hares, [99–101](#)
sensory modality regaining through perceptual learning, [114–116](#)
to stress in anxiety and depression, [112–114](#)

Sensory cortex, [301](#)

Sensory deprivation, [36](#)

Sensory memory, [371–372](#)

Sensory preconditioning, [244–245](#), [263–264](#), [264f](#)

Sensory prosthesis, [114–115](#), [363](#)

Sensory stimuli, [257–260](#)

Serial reaction time task, [338](#)

Sex, as conditioned or unconditioned stimulus, [133t](#)

- Sex hormones on brain organization, [530–532](#)
- Sexual conditioning, [126](#), [126f](#)
- Sexual habituation experiment, [80](#)
- Shaping, [182–183](#)
- Shared-elements models of receptive fields, [258–260](#)
- Short-term memory, [370](#), [372–374](#)
 - behavioral distinctions between long-term memory and, [372t](#)
 - controlled updating of buffers in, [381–384](#)
- Sign language, [511](#), [512](#)
- Simple forgetting, [291–292](#), [292f](#)
- Single-cell recording, [53](#), [54f](#)
- Skill decay, [346](#)
- Skill memory, [328–367](#)
 - in animals, [333–334](#)
 - artificial limbs and, [363–365](#)
 - basal ganglia and skill learning, [348–354](#)
 - behavioral processes and, [330–347](#)
 - brain substrates and, [348–362](#)
 - cerebellum and timing, [358–362](#)
 - clinical perspectives on, [362–365](#)
 - cognitive vs. perceptual-motor, [331–332](#)
 - cortical representations in, [354–356](#)
 - features of, [330–334](#)
 - memory encoding, [334–343](#)
 - memory failure, [345–347](#)
 - Parkinson’s disease, [362–363](#)
 - retrieving existing memories, [344–348](#)
 - storage of, [356–358](#)
 - talent and, [339–341](#)
 - unconsciously forming, [337–339](#)
- Skills
 - brain substrates in learning, [348–354](#)
 - closed, [331](#), [332](#)
 - cortical representations of, [354–356](#)
 - defined, [330](#)
 - open, [331](#), [332](#)

Skin conductance response (SCR), [441–442](#), [442f](#)

Skinner, B. F., [13–14](#), [13f](#), [24](#), [31t](#), [37](#)

Skinner box, [179–180](#), [180f](#)

Sleep

- forgetting and, [36](#)
- learning during, [317–318](#)
- memory and, [315–318](#)
- stimulus control and, [240](#)

Slow-wave sleep (SWS), [315–316](#)

Social conformity, [485–486](#)

Social learning, defined, [468](#)

Social learning and memory, [466–499](#). *See also* [Copying](#)

- autism spectrum disorder (autism), [494–495](#)
- behavioral processes and, [468–488](#)
- brain substrates and, [489–492](#)
- clinical perspectives on, [492–495](#)
- of food preferences, [484–485](#)
- imitative deficits after stroke, [492–493](#), [493f](#)
- mirror neurons, [489–492](#)
- social transmission of information, [483–488](#)
- song learning in bird brains, [491–492](#), [491f](#)

Social learning theory, [470–472](#)

Social transmission of information, [483–488](#)

- learning through social conformity and, [483–486](#)
- media's impact on behavior, [486–488](#), [487f](#)

Socrates, [1](#)

Soma, [41](#)

Somatic theories of emotion, [422](#)

Somatosensory cortex, [102–103](#), [103f](#)

Song learning, [481–483](#), [491–492](#), [491f](#)

Source monitoring error, [294](#)

Spaced exposure, [80](#)

Spaced practice, [336](#)

Spanking, [189](#)

Spatial contiguity, [1–2](#)

Spatial learning, [93–95](#)

Specificity

generalization and, [79](#)

learned, [239–242](#)

transfer, [344](#)

Spines, [527–528](#)

Spontaneous recovery, [81](#)

Standard consolidation theory, [313](#)

Statistical learning, [90](#). *See also* [Perceptual learning](#)

Stellate cells, [41](#)

Stereotype

accuracy of, [253–254](#)

defined, [252](#)

interpreting and using, [253–254](#)

learning of, [252–253](#)

Stimulation, deep brain, [363](#), [363f](#)

Stimulus, [6](#), [18–19](#), [19f](#)

attentional approach to, [146–149](#)

attentional explanation of latent inhibition, [147–148](#)

attention and modulation of conditioned, [146–149](#)

classical conditioning, [146–149](#)

conditioned, [123–125](#)

discriminative, [177](#), [181](#)

enhancement, [477–478](#)

incoming, [49](#), [49f](#)

motivational value of a, [209–210](#)

overselectivity, in autism, [271–272](#)

representation, [232](#)

selection and response inhibition, [386–388](#)

Stimulus control, [238–243](#)

defined, [238](#)

discrimination learning and, [238–243](#)

errorless discrimination learning and easy-to-hard transfer, [242–243](#)

peak shifts in generalization, [229–230](#)

sleeping better through, [240](#)

Stimulus–response models, [23–26](#), [25f](#)

Stimulus sampling theory, [26](#)

Storage of episodic memory, [445–446](#)

Stress, [420](#)

- forgetting and, [36](#)

Stress hormones, [420](#)

- memory encoding and, [448–451](#)

Stroke, [111–112](#), [492–493](#), [493f](#)

Stroop task, [387](#)

Structural neuroimaging, [40](#)

Study habits, optimizing, [300](#)

Subcortical structures involved in episodic and semantic memory, [310–311](#)

Subject bias, [21](#)

Subsequent memory paradigm, [306–307](#), [448](#)

Substantia nigra pars compacta (SNc), [209f](#), [210](#)

Super Bowl ads, [488](#)

Supervisory attentional system, [387](#)

Synapse, [60–62](#)

- brain functions, [60–62](#), [61f](#)
- defined, [61](#)
- in learning in *Aplysia*, [97](#)
- localized neuron and, [533–535](#), [534f](#)
- long-term structural changes and creation of new, [164–165](#)
- loss, [533–535](#), [534f](#)
- plasticity of, [62](#), [64](#)
- transmission of, [60–62](#), [61f](#)

Synaptic depression, [98](#)

Synaptic plasticity, [62](#)

Synaptic stability, [535–536](#)

Synaptogenesis, [527](#)

Systematic desensitization, [455–456](#)

T

Talent, [341–343](#)

Tangles in brain, [542–544](#)

Targeted memory reactivation, [317](#)

Task

- categorization, [145f](#), [376](#)

- delayed non-match-to-sample, [377–378](#)

- novel object recognition, [87](#)

- rotary pursuit, [341–342](#), [342f](#)

- switching, [385–386](#)

- weather prediction, [353–354](#), [353f](#)

- word-stem completion, [88](#)

- working memory, [395–398](#)

Taste aversion, conditioned, [151–152](#)

Template model of song learning, [482](#), [482f](#)

Temporal contiguity, [1–2](#), [190–191](#)

Temporal lobe, [38](#)

Tend-and-befriend response, [426–427](#)

Terrorism, [417](#)

Testing effect, [289–291](#)

Testosterone, [515](#), [530–531](#)

Tetraplegia, [364](#)

Thalamus, [39](#), [39f](#)

- emotional influences on memory and, [439](#)

Theory, [1](#)

- of emotion, [421–424](#), [421f](#)

- of equipotentiality, [59](#)

- of natural selection associated with learning and memory, [15–17](#), [17t](#)

Thorndike, Edward, [8](#), [8f](#), [31t](#)

Timing

- cerebellum and, [358–362](#)

- classical conditioning, [150–151](#)

- and sequence for memory encoding, [336–337](#)

Tip-of-the-tongue (TOT) phenomenon, [299](#), [519](#)

Token economies, [186](#)

Tolerance, [131–132](#)

Tolman, Edward, [17–19](#), [18f](#), [31t](#)

Topographic organization in generalization, [260–261](#)

Tower of Hanoi puzzle, [385](#), [385f](#)

Trace conditioning, [150–151](#), [150f](#), [505](#)
 trace forms of eyeblink conditioning, [150–151](#), [150f](#)

Training
 on-the-job, [44–45](#)
 transfer of, [344](#), [351](#)

Transcranial direct current stimulation (tDCS), [67–68](#), [298](#)

Transcranial magnetic stimulation (TMS), [67–68](#), [67f](#)

Transfer-appropriate processing (TAP) effect, [288–289](#)

Transfer of training, [344](#), [351](#)

Transfer specificity, [344](#)

Transient global amnesia (TGA), [320–321](#)

Transient memories, [370–374](#)
 defined, [370](#)
 sensory memory and, [371–372](#)
 short-term memory, [372–374](#)

Trial-level model, [150–151](#)

Trisomy [21](#), [539](#)

True imitation, [472–474](#)

Twin studies, [521–523](#), [522f](#)

Two-action test, [472–473](#)

Two-factor theory of avoidance learning, [431–432](#), [431f](#)

Two-factor theory of emotion, [421f](#), [422–424](#)

U

Unconditioned response (UR), [123](#)

Unconditioned stimulus (US), [123–125](#)

 associative weights and compound conditioning, [140](#)

 classical conditioning, [135–146](#)

 cue-outcome contingency and judgments of causality, [144–145](#)

 cues for, [132–133](#)

 error-correction learning, [139–140](#)

 in human category learning, [143–146](#)

 informational value of cues, [135–136](#)

 Kamin's blocking effect, [136–138](#)

 modulation theory, [148](#)

 Rescorla–Wagner model of conditioning, [138](#), [140t](#)

 sex as, [133t](#)

Unconscious racial bias, [91](#)

V

Val allele (of *BDNF* gene), [521](#)

Variable-interval schedules, [196–197](#)

Variable practice, [337](#)

Variable-ratio (VR) schedule, [195f](#), [196](#)

Ventral tegmental area (VTA), [209](#), [209f](#)

Ventrolateral prefrontal cortex (VLPFC), [394](#), [394f](#)

Video games, [352](#), [486–487](#)

Virtual-reality therapy, [455–456](#)

Vision, sensitive period for development of, [509–510](#)

Visual landmarks, [107–108](#), [108f](#)

Visual sensory memory, [371](#)

Visuospatial sketchpad, [374](#)

Vocal imitation, [479–480](#)

Vocal learning, [481](#)

W

“Wanting” and “liking” in the brain, [209–213](#), [209f](#), [211f](#)

addiction and, [216–219](#)

Watson, John, [11–13](#), [12f](#), [31t](#), [36–37](#), [454](#)

Wearing, Clive, [21](#), [22](#)

Weather prediction task, [353–354](#), [353f](#)

Weight, associative, [140](#)

Who’s Who in the history of learning and memory, [23](#)

Wisconsin Card Sorting Test, [386](#), [386f](#), [393–394](#), [407](#), [409](#)

Word-length effect, [376](#)

Word-stem completion task, [88](#)

Working memory, [368–415](#)

in aging adults, [516–517](#)

attention deficit/hyperactivity disorder, [410–412](#)

Baddeley’s model of, [374–375](#)

behavioral processes and, [370–391](#)

brain substrates and, [391–407](#)

clinical perspectives on, [407–412](#)

cognitive control, [381–392](#)

defined, [370](#)

frontal brain activity during working-memory tasks, [395–398](#)

frontal lobes and consequences of damage to, [391–394](#)

intelligence, cognitive control, and, [388–389](#)

mapping onto prefrontal cortex, [398–403](#)

maturation of in adolescence, [513–514](#)

phonological loop and, [374](#), [375–377](#)

place vs. state of, [378–380](#)

prefrontal control of long-term declarative memory, [403–407](#)

taking breaks for, [390](#)

tasks, [395–398](#)

transient memories, [370–372](#)

visuospatial sketchpad and, [376–378](#)

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